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EFFECT OF ESTROGENIC HORMONE ON ADVANCED CARCINOMA OF THE FEMALE BREAST

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THE FAVORABLE results obtained by a group of English investigators [1] from the use of estrogenic hormones in some cases of advanced carcinoma of the female breast prompted us to try this therapeutic procedure. We selected 17 patients for study. Thirteen patients had primary inoperable cancer, for which no previous therapy had been administered. Two patients had recurrent lesions in the same breast several years after treatment of inoperable carcinoma by irradiation. Of the remaining 2 patients of this group, a carcinomatous ulcer had developed in the mastectomy scar of 1 and recurrent nodules in the skin of the thoracic wall of the other. A biopsy specimen which was positive for cancer was obtained in every case.

These 17 patients were treated exclusively with a synthetic estrogenic hormone (ethinyl estradiol) administered orally. One milligram of this substance, according to some investigators,[2] is the equivalent of 7 to 20 mg. of diethylstilbestrol. In the present investigation the dose of ethinyl estradiol varied from 0.15 to 0.7 mg. daily. On the basis of the lower figure given previously, this would be equivalent to at least 1.0 to 5.0 mg. of diethylstilbestrol daily.

The data in connection with these 17 patients have been tabulated (table). The ages of these women ranged from 40 to 77 years. Five patients were younger than 60 years. The 40 year old patient was the only one who was not in the postmenopause period. The duration of the estrogen treatment varied from two to eleven months.

From the Breast Department of the Memorial Hospital for the Treatment of Cancer and Allied Diseases.

1. (*a*) Discussion on Advanced Cases of Carcinoma of the Breast Treated by Stilbestrol, Proc. Roy. Soc. Med. **37**:731, 1944. (*b*) Haddow, A.; Watkinson, J., and Paterson, E.: Influence of Synthetic Oestrogens upon Advanced Malignant Disease, Brit. M. J. **2**:393, 1944.

2. Soule, S.: A Clinical Trial of Ethinyl Estradiol, Am. J. Obst. & Gynec. **45**:315, 1943. Allen, W.: The Biological Activity of Various Estrogens, South. M. J. **37**:270, 1944.

Summary of Seventeen Cases Treated by Estrogenic Hormone

Case	Age, Yr.	Status Before Estrogen Therapy	Average Daily Dose, Mg.	Total Dose, Mg.	Duration of Therapy	Course
1	62	Mass in left breast and axilla; bilateral supraclavicular nodes; pulmonary metastases; postmenopause	0.3	12.6	6 wk.	No change in lesions; died suddenly of coronary occlusion 6 wk. after institution of therapy
2	56	Radical mastectomy on right side 5 yr. previously; mass in left breast, axilla and supraclavicular space; postmenopause	0.25	47.25	6 mo.	Mass increased until whole left breast was involved; nodules developed in breast skin; uterine bleeding induced; serial histologic specimens revealed cytologic changes; died 12 mo. after institution of estrogen therapy
3	40	Mass occupying entire right breast; bilateral axillary and right supraclavicular nodes; menstruating regularly	0.25	19.25	11 wk.	En cuirasse phenomenon developed in right breast; metastasis to left breast and lungs; died 4 mo. after institution of hormone therapy
4	57	Mass in left breast, axilla and supraclavicular space; postmenopause	0.25	42.0	6 mo.	Mass in breast and axilla increased in size; metastasis to right axilla developed; uterine bleeding induced; died 8 mo. after institution of hormone therapy
5	58	Mass occupying whole right breast; cutaneous nodules; right axillary and left supraclavicular nodes; postmenopause	0.25	31.5	5 mo.	En cuirasse phenomenon developed in right breast; pulmonary metastasis; uterine bleeding induced; died 16 mo. after institution of hormone therapy
6	55	Fungating mass involving one third of breast; extensive pulmonary metastases; postmenopause	0.25	57.75	11 mo.	Increase in size and extent of fungating mass; decrease in number, density and extent of pulmonary metastases; uterine bleeding induced; specimens for serial biopsies revealed no change; died 18 mo. after institution of hormone therapy
7	75	Entire left breast involved; bilateral axillary nodes; edema of the left arm; postmenopause	0.3	50.4	6 mo.	Nodules in skin of left breast and edema of legs developed; no uterine bleeding; specimens for serial biopsies revealed no change; died 7 mo. after institution of hormone therapy
8	69	Ulcer in mastectomy scar on right side; hard mass in right axilla; numerous skin nodules; postmenopause	0.15	9.45	4 mo.	Base of ulcer on thoracic wall healed; biopsy specimens revealed no cytologic change; uterine bleeding induced; general condition good 9 mo. after institution of estrogen therapy
9	61	Ulcerated mass in upper outer quadrant of left breast; metastasis to spine, pelvis, ribs, femurs and skull; paralysis of the right side of the face; postmenopause	0.30	16.8	2 mo.	Grew progressively worse; repeat roentgenograms revealed increase in extent of metastases; uterine bleeding induced; specimens for serial biopsies revealed no change; died 6 mo. after first clinic visit

10	68	Nodular mass in left breast; skin red, impending ulceration; a few 1 cm. lymph nodes in left axilla; postmenopause	0.45	50.4	4 mo.	Area of softening became smaller; gained 8 pounds in weight; active uterine bleeding induced; general condition good 7 mo. after institution of estrogen therapy
11	63	Right breast and overlying skin completely infiltrated with cancer; axillary mass on right side; edema of the right arm; pulmonary metastases; postmenopause	0.5	26.6	2 mo.	Repeat roentgenograms of chest after 2 mo. of estrogen treatment revealed diminution in size of metastases; breast lesion unchanged; no uterine bleeding; specimen for serial biopsies revealed no change; general condition is good 4 mo. after institution of estrogen therapy
12	60	Mass protruding from left breast; ulceration imminent; firm nodes in left axilla; metastasis to lungs and pelvis	0.6	33.6	2 mo.	Mass in left breast decreased in size; general condition good 4 mo. after institution of estrogen therapy; no uterine bleeding
13	73	Mass in left breast, with small area of ulceration; postmenopause	0.5	28.0	2 mo.	Ulceration healed completely; uterine bleeding induced; specimens for serial biopsies revealed no change; condition good 4 mo. after institution of estrogen therapy
14	71	Left breast and skin widely infiltrated with carcinoma; postmenopause	0.5	17.5	5 wk.	Uterine bleeding developed; not able to return to clinic; specimens for serial biopsies failed to reveal cytologic changes
15	77	Left breast shrunken, filled with hard mass, large ulceration; 2 cm. node in left axilla; postmenopause	0.5	42.0	3 mo.	Ulceration filled in; mass grew smaller; axillary node disappeared; no uterine bleeding; specimens for serial biopsies revealed no change; in good condition 4 mo. after institution of estrogen therapy
16	62	Fungating masses occupying most of right breast; left breast involved by inflammatory carcinoma extending onto abdomen; numerous bilateral supraclavicular nodes; postmenopause	0.5	26.0	2 mo.	Local condition increased in extent; general condition good 2 mo. after institution of estrogen therapy; no uterine bleeding
17	74	Simple mastectomy 8/20/45; recurrent skin nodules 3/4/46; postmenopause	0.7	35.2	2 mo.	Condition essentially unchanged 2 mo. after institution of estrogen therapy

RESULTS

In this group of 17 patients 7 died of the disease from four to eighteen months after the institution of estrogen therapy. One died of coronary occlusion after six weeks of treatment. Five of the deceased patients were less than 60 years of age. Only 1 of the latter group (case 6) manifested a transient favorable response to the therapy. One patient, in poor condition when first seen at this clinic, was unable to return after five weeks of therapy because her condition rapidly deteriorated.

Seven of the total number of patients, or 40 per cent, exhibited some clinical improvement. Preexistent carcinomatous ulcers became smaller in cases 8 and 15 and healed completely in case 13. The changes produced in case 15 are shown in figure 1. In case 12 a protuberant nonulcerated cancer mass decreased in size (fig. 2). A diminution in the size of the breast mass and a solidification of an area of softening were exhibited in case 10. A decrease in the size and extent of pulmonary metastatic lesions was revealed in cases 6 and 11. This is shown in figures 3 and 4. The fungating lesion arising from the breast of the patient in case 6 continued to increase in size during the period of regression of the pulmonary lesions. This patient died eighteen months after the institution of estrogen therapy. The other 6 patients in this group are in apparent good condition. The disease has regressed in each instance, but in no case to date has it entirely disappeared. These patients have been under observation for periods ranging from four to nine months.

Although there was no clinical evidence of improvement in case 2, specimens for serial biopsies taken during the course of the treatment revealed cytologic changes. One biopsy specimen taken after five months of therapy was described by Dr. Fred Stewart as follows: "Mammary carcinoma infiltrating dermal lymphatic vessels and blood vessels is present. The appearance is slightly abnormal in that the nuclei look unusually hyperchromatic and irregular. The cytoplasm is unusually abundant and a little granular. Mitoses are abundant. There is nothing peculiar in the original tumor; hence these changes are definite."

The patients that responded favorably manifested the improvement within the first four to six weeks. It appeared that if favorable results were not obtained relatively early the continued use of the hormone would be ineffectual. Most of the women treated experienced a feeling of well-being while under estrogen therapy. A few gained in weight. One patient (case 10) gained 8 pounds (3.6 Kg.). Six of the 7 patients in whom favorable changes were produced by the hormone were over 60 years old.

The hormone induced uterine bleeding in 8 patients during the course of the treatment. On 1 occasion a severe hemorrhage occurred.

Fig. 1 (case 15).—*A*, the left breast is seen to be filled with a nodular mass, above which is a large area of ulceration and lateral to this a smaller area of impending ulceration. *B*, this photograph was taken after two months of estrogen therapy. The mass in the breast has regressed decidedly. The ulcerated area has filled in and the area of impending ulceration has disappeared.

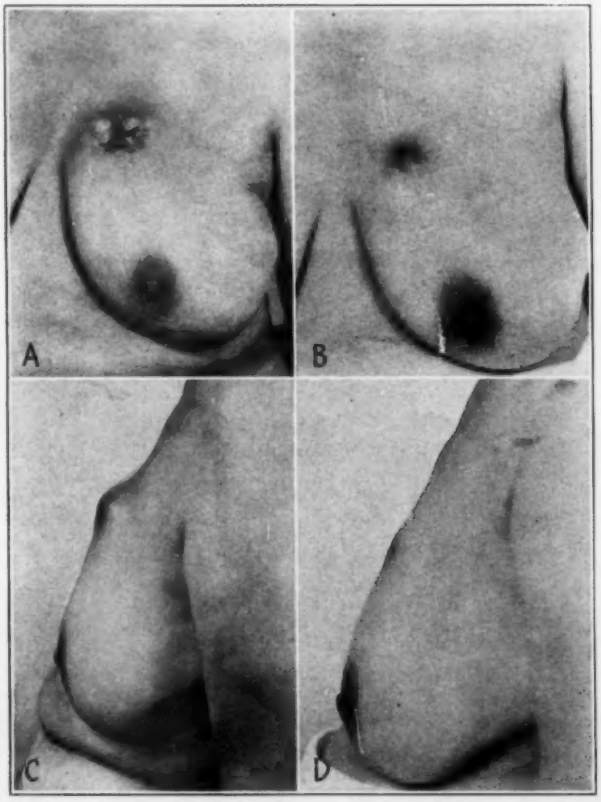

Fig. 2 (case 12).—*A*, there is an area of impending ulceration at the medial aspect of the lesion. *B*, after two months of estrogen therapy the area of impending ulceration has disappeared. *C*, this is a profile view of the protuberant lesion shown in *A* before estrogen therapy was initiated. *D*, this is the profile view of the protuberant lesion after two months of estrogen therapy. A comparison with *C* reveals a striking regression of the lesion.

After two months of estrogen therapy vaginal smears [3] were made in 6 patients (cases 11, 12, 13, 15, 16 and 17), the technic of Shorr [4] and of Papanicolaou [5] being followed. The cytologic changes associated with estrus which have been described by these investigators were present in each instance. In some of the women in the sixth and seventh decades the breasts became fuller and the overlying skin pink.

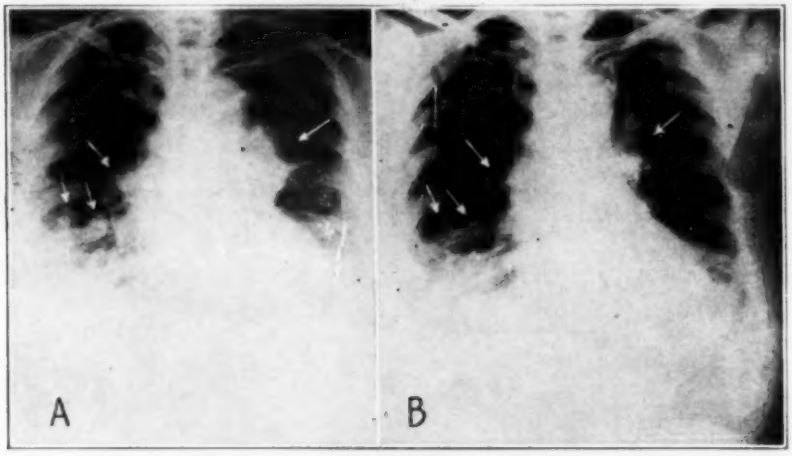

Fig. 3 (case 6).—*A*, extensive pulmonary metastases may be seen. *B*, this roentgenogram was made after three months of estrogen therapy. The regression of the metastatic lesions may be seen by comparison with the preceding figure.

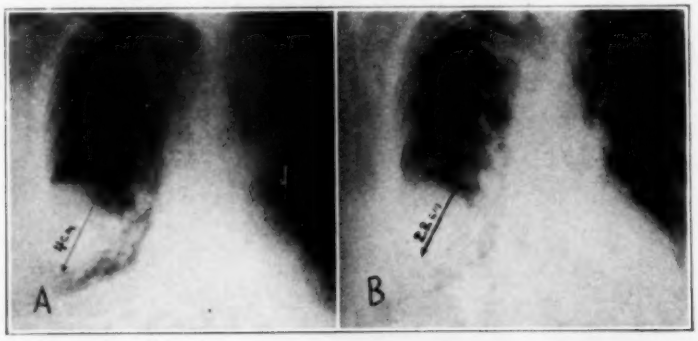

Fig. 4 (case 11).—*A*, there is a large metastatic area near the base of the right lung. *B*, this roentgenogram, taken after two months of estrogen therapy, shows the lesion at the right base to have decreased in size by almost one half.

3. Dr. Anne C. Carter, of the Department of Endocrinology, New York Hospital, interpreted these smears.

4. Shorr, E.: An Evaluation of the Clinical Application of the Vaginal Smear, J. Mt. Sinai Hosp. **12**:667, 1945.

5. Papanicolaou, G.: The Sexual Cycle in the Human Female as Revealed by Vaginal Smears, Am. J. Anat. (supp.) **52**:519, 1933.

Mild toxic reactions, which were evidenced by abdominal cramps and nausea, were experienced by most of the patients in the early stages of treatment, even with relatively small doses of the estrogen. A tolerance was quickly established, however, so that the dose could be increased rapidly. In 1 patient (case 7) edema of the legs and ankles developed, which was in all probability a toxic manifestation of the estrogen.

Monthly determinations of the serum calcium, phosphorus and alkaline phosphatase levels were made in all patients except 3 (cases 1, 14 and 17). In no case was there definite evidence that an alteration in the serum calcium or alkaline phosphatase contents was induced by estrogen therapy. There was a significant drop in the serum phosphorus level in 6 patients. In the remainder no appreciable change was noted. It is of interest that Buchwald and Hudson,[6] working with male rats, found that a subcutaneous daily injection of diethylstilbestrol did not influence the serum calcium level but did produce an apparent decrease in the serum phosphorus content.

There were no significant changes noted in routine examinations of the blood and urine except in 1 patient (case 11). This patient had sugar (3 plus) in her urine at her initial visit to the clinic. A determination of blood sugar content revealed a hyperglycemia of 276 mg. per hundred cubic centimeters. The patient had suffered from polyuria and nocturia for many months but was unaware of the cause. She had never received treatment, nor had she ever been on a low carbohydrate diet. She was asked to continue with her usual diet. No insulin was given. After two weeks of estrogen therapy her blood sugar content was 206 mg. per hundred cubic centimeters, and at the end of six weeks of estrogen treatment it was 140 mg. Her urine at this time was sugar free, and the urinary symptoms had disappeared. She was eating carbohydrates in abundance.

COMMENT

The only group of patients with advanced carcinoma of the breast treated by estrogens reported to date is that of the English investigators.[1a] In that symposium ten physicians pooled their cases, which totaled 100. Our findings do not differ essentially from theirs. An important point of agreement is that the favorable results are obtained predominantly in women above the age of 60.

The results of the present investigation suggest that estrogens have a decidedly deleterious effect on women in the younger age group.

6. Buchwald, K., and Hudson, L.: The Biochemical Effects of Sex Hormones, Acid and Alkaline Phosphatase Activity, Calcium and Phosphorus, Endocrinology **35**:73, 1944.

This appeared to be most striking in the 40 year old patient (case 3), who was menstruating regularly. When first seen in the clinic her general condition was good despite the mass in the right breast and right axilla. Roentgenograms revealed no evidence of disease in the lungs, lumbar portion of the spine or pelvis. Soon after the institution of the estrogen therapy her condition deteriorated. Within three months she was bedridden, and she died about one month later. A comparison of *A* and *B* in figure 5 reveals the progress of the disease in ten weeks. In figure 5 *B* the opposite breast is seen to be involved. This breast is edematous, and the nipple is retracted. The rapid progress of the disease may have been the result of a specific action of the estrogen, or it may have resulted from vascular changes produced in the breast by the hormone.

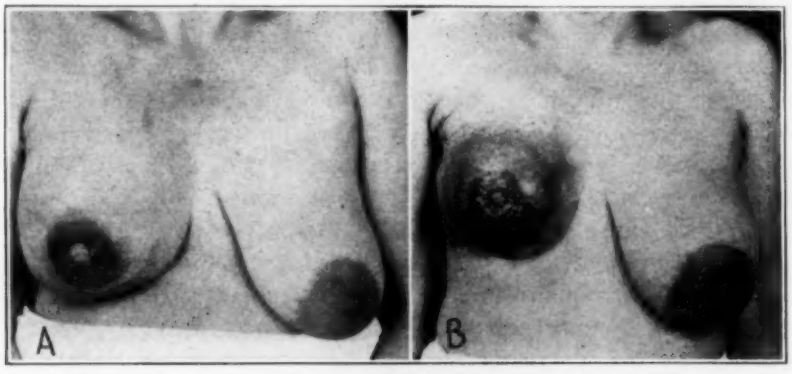

Fig. 5 (case 3).—*A*, this is a photograph of the breasts before estrogen therapy was instituted. The deformity of the right breast, retraction of the nipple and pigskin appearance are clearly delineated. The left breast is apparently normal. *B*, this photograph was taken after ten weeks of estrogen therapy. Comparison with *A* reveals the striking change that has taken place in the right breast and also the edema, retraction of the nipple and pigskin appearance now present in the left breast.

Both estrogens and androgens [7] apparently exert an inhibitory effect on carcinoma of the female breast. The pituitary gland may be the common denominator. Moore and Price [8] have presented evidence that the pituitary may be influenced by the sex hormones. They found that gonadal hormones of either sex exert a depressing effect on the hypophysis of male rats, which results in a diminished amount of the

7. Adair, F., and Herrmann, J.: The Use of Testosterone Propionate in Advanced Carcinoma of the Breast, Ann. Surg. **123**:1023, 1946. Herrmann, J.; Adair, F., and Woodard, H.: Further Experience with Testosterone Propionate in Osseous Metastasis from Carcinoma of the Female Breast, to be published.

8. Moore, C., and Price, D.: Gonad Hormone Functions and the Reciprocal Influence Between Gonads and Hypophysis with Its Bearing on the Problem of Sex Hormone Antagonism, Am. J. Anat. **50**:13, 1932.

sex-stimulating factor available to the organism. Badger[9] and his co-workers concluded from their researches that some synthetic estrogens exert inhibition of growth on animal neoplasms. In their opinion the action might be in part local on the tumor cells and in part central on the pituitary.

SUMMARY AND CONCLUSIONS

1. The results obtained from treating 17 women with advanced carcinoma of the breast with estrogenic hormone are reported.

2. There was a favorable response noted in 7, or 40 per cent, of the patients, 5 with superficial lesions of the soft parts and 2 with pulmonary metastases.

3. The favorable response occurred predominantly in women over 60 years.

4. The skin of the breasts in elderly women may become pink and the mammae engorged.

5. The dosages employed induced uterine bleeding, sometimes severe, in over 50 per cent of the patients.

6. Vaginal smears were studied in 6 patients, all of whom showed a well developed estrus reaction.

7. There is suggestive evidence that in young women estrogenic hormone may accelerate the progress of the disease.

8. There were no changes in serum calcium, protein or alkaline phosphatase levels that could be ascribed to the administration of daily doses of an estrogen (ethinyl estradiol) the equivalent of 3 mg. of diethylstilbestrol. A fall in the serum phosphorus level was observed in a large percentage of cases.

9. Hyperglycemia was decidedly reduced in 1 patient solely by the use of estrogen.

The Schering Company, of Bloomfield, N. J., supplied the synthetic estrogen ethinyl estradiol employed in this investigation.

9. Badger, G.; Elson, L.; Haddow, A.; Hewett, C., and Robinson, A.: The Inhibition of Growth By Chemical Compounds, Proc. Roy. Soc., London, s. B **130**: 255, 1942.

PATENT OMPHALOMESENTERIC DUCT AND ITS RELATION TO THE DIVERTICULUM OF MECKEL

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MECKEL'S diverticulum and the patent omphalomesenteric duct are the consequence of the same defect in embryologic development, differing only in the extent to which the omphalomesenteric duct fails to obliterate. Although Meckel's diverticulum is relatively common, with a frequency of 1 to 2 per cent,[1] the patent omphalomesenteric, or vitelline, duct is a rare anomaly. Two patients with complete patency of the omphalomesenteric duct have been treated at the University of Chicago Clinics and were the only patients with this condition in over 30,000 births at the Chicago Lying-In Hospital of the University of Chicago during the past ten years. A review of the literature reveals 128 reported cases. An additional case has been observed elsewhere,[2] bringing the total number of cases of patent omphalomesenteric, or vitelline, duct to 131. The most important and serious aspect of this anomaly is the complication of prolapse of the intestine externally through the umbilical fistula, which occurred in 27 of the reported cases, including 1 of ours.

HISTORICAL DATA

One of the earliest known descriptions of an intestinal diverticulum was made by the German surgeon and anatomist Fabricius Hildanus

From the Department of Surgery, University of Chicago, The School of Medicine.

This work was aided by a grant from the Douglas Smith Foundation for Medical Research, of the University of Chicago.

1. (*a*) Christie, A.: Meckel's Diverticulum: A Pathologic Study of Sixty-Three Cases, Am. J. Dis. Child. **42**:544 (Sept.) 1931. (*b*) Cullen, T. S.: Embryology, Anatomy, and Diseases of the Umbilicus Together with Diseases of the Urachus, Philadelphia, W. B. Saunders Company, 1916. (*c*) Griffith, J. P.: Diseases Connected with Meckel's Diverticulum with Especial Reference to Diverticulitis, J. A. M. A. **62**:1624 (May 23) 1914. (*d*) Harbin, R. M.: Meckel's Diverticulum, Surg., Gynec. & Obst. **51**:863, 1930. (*e*) Lichtenstein, M. E.: Meckel's Diverticulum, Quart. Bull., Northwestern Univ. M. School **15**:296, 1941.

2. Dr. J. E. Morgan, Cleveland.

in 1598. A similar case was reported by Lavater in 1672. Fredericus Ruysch is credited with publishing the first known picture of an ileal diverticulum, which he termed the "preternatural diverticulum." Early clinicians attributed the origin of the diverticulum to mechanical forces. In 1700 Littre described a patient with a hernia which contained a diverticulum of the ileum, and his name has since been an eponym for this condition. Other cases of diverticulum found in a hernia were reported by Mery and by von Taignon [3] in 1701. The first cases of ileus due to diverticulum of the ileum were described by van Doeveren, Duvignaud, Monro and Moscati [3] in the latter half of the eighteenth century. Morgagni, an Italian pathologist and anatomist, considered a congenital origin for this diverticulum, which he called "true" in contrast to diverticula of the rectum, which were "false" because they consisted of only one intestinal layer instead of all layers. Bose in 1779 demonstrated that the ileal diverticulum was congenital in origin. Luca interposed an amusing theory when he postulated that the intestine was formed by growth from the two ends of the embryo uniting in the middle. If malunion and overlapping occurred, a diverticulum was formed.

It remained for Johann Frederic Meckel to clarify the origin and significance of the diverticulum which bears his name today. In a series of three papers published between 1808 and 1815, he established the following points: The diverticulum was solitary, antimesenteric and congenital and located in the ileum at the site where the original yolk stalk coursed between the gut and yolk sac. He explained the condition as incomplete disappearance of the omphalomesenteric duct. Observing obliterated umbilical vessels persisting as fibrous strands between the umbilicus and the diverticulum, he considered these as support for his theory of origin. Since the early work of Meckel, his explanation of its origin has been confirmed and its importance in the causation of abdominal pathologic conditions has been stressed.

EMBRYOLOGY

An understanding of the embryologic background of Meckel's diverticulum and the patent omphalomesenteric duct is necessary to appreciate fully these anomalies and to distinguish them from other conditions important in differential diagnosis.

The embryologic development of the umbilical region is well presented by Cullen.[1b] The formation of the exocelom separates the ento-

3. Neff, G.: Das Meckelsche Divertikel, Ergebn. d. Chir. u. Orthop. **30**:227, 1937.

derm from the periphery of the ovum, and the yolk, or vitelline, sac is formed (fig. 1*a* and *b*). In this and subsequent stages the yolk, or omphalomesenteric, duct forms a direct communication between the primitive gut and the umbilical vesicle or yolk sac. Its blood supply comes from two omphalomesenteric arteries, branches of the aorta, and the return circulation is carried through two omphalomesenteric veins, tributaries to the future portal circulation. The allantois originally communicates with the yolk sac. With growth of the fore-

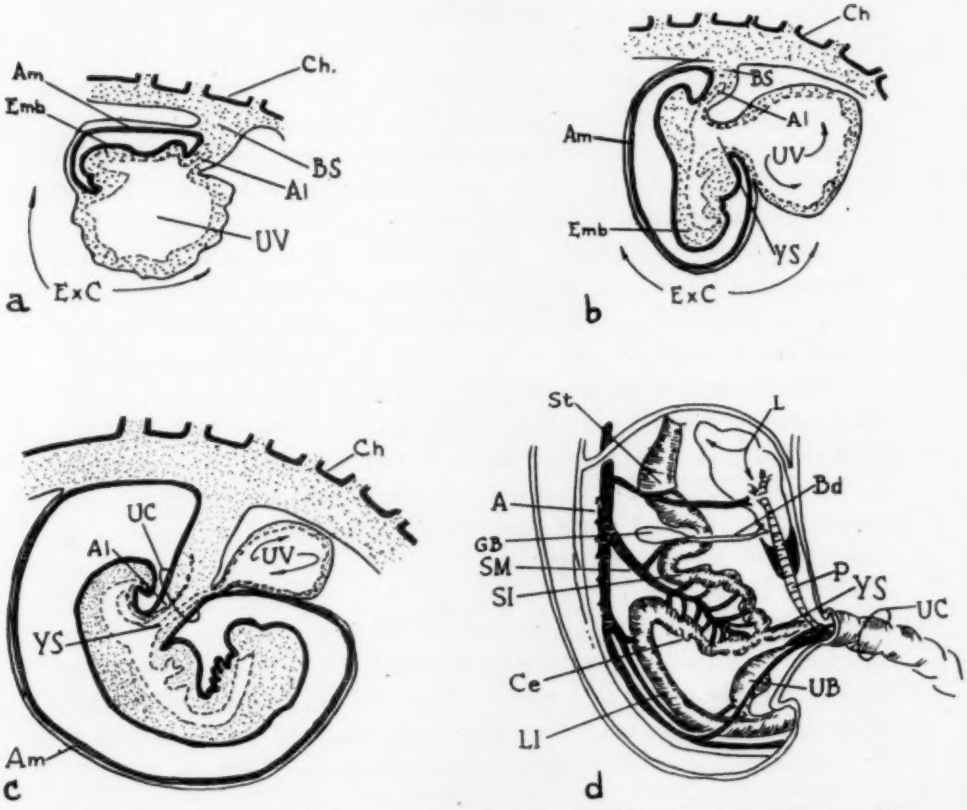

Fig. 1.—The embryologic development of the umbilical region, with especial reference to the vitelline, or omphalomesenteric, duct. *A* indicates the aorta; *Al*, allantois; *Am*, amnion; *Bd*, bile ducts; *BS*, body stalk; *Ce*, cecum; *Ch*, chorion; *Emb*, embryo; *ExC*, extracoelomic cavity; *GB*, gallbladder; *L*, liver; *LI*, large intestine; *P*, portal vein; *SI*, small intestine; *SM*, superior mesenteric artery; *St*, stomach; *UB*, urinary bladder; *UC*, umbilical cord; *UV*, umbilical vesicle, and *YS*, yolk stalk.

parts and hindparts of the embryo, especially the former, the communication to the yolk sac becomes narrowed and is brought into juxtaposition with the body stalk, so that the structures in this vicinity become grouped together and are termed the umbilical cord (fig. 1 *c* and

d). Passing from the convexity of the first loop of bowel, the vitelline duct is visible at this stage. During embryonic development the intestine extrudes transiently into the umbilical stalk and later withdraws into the celomic cavity. The vitelline duct normally undergoes obliteration during the sixth or seventh week of embryonic life. Obliteration of none or only a part of this duct leads to a number of anomalies, the commonest of which is the type of diverticulum originally described by Meckel.

Other structures developing in the umbilical region should be mentioned because of their importance in diagnosis. The allantoic diverticulum forms the primitive urinary bladder early in embryonic life. The urachus within the umbilical cord eventually joins the urinary bladder. The time of urachal obliteration is variable. According to Cullen, the urachus has a tendency toward dilation at irregular intervals. The development of the urinary bladder, the fate of the allantois and the role of the urachus are still controversial.[4]

The anomalous development of structures of the umbilical cord exists in many varied forms, depending on the extent to which obliteration occurs in the omphalomesenteric duct, the urachus and the vitelline vessels, separately and in combinations. The commonest anomaly is the one in which the proximal portion of the duct fails to undergo obliteration, producing the usual type of Meckel's diverticulum (fig. 2*A*). This diverticulum may contain heterotopic tissue, such as gastric mucosa, with the possibility of ulcer formation. A band of fibrous tissue may lead from the umbilicus to the diverticulum, representing remnants of the omphalomesenteric duct or the vitelline vessels[5] (fig. 2*B*). The fibrous band may attach to nearby structures (fig. 2*C* and *D*).

When the proximal portion of the duct obliterates normally while the distal portion fails to disappear, a number of conditions may arise. A mucosal-lined polypoid lesion of the umbilicus secreting a mucous material (fig. 2*E*) may occur. There may be a fibrous band leading from this to the ileum (fig. 2*F*). The most distal portion of the duct may form a cyst beneath the umbilicus (fig. 2*G*), or there may be a fibrous cord leading from such a persistent distal end to the intestine (fig. 2*H*).

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4. Begg, R. C.: The Urachus and Umbilical Fistulae, Surg., Gynec. & Obst. **45**:165, 1927. Hammond, G.; Yglesias, L., and Davis, J. E.: Urachus: Its Anatomy and Associated Fascia, Anat. Rec. **80**:271, 1941.

5. (*a*) Fraser, J., and McCartney, J. E.: A Case of Persistent Vitelline Artery: Fatal Intra-Abdominal Hemorrhage from Rupture of the Vessel, Brit. J. Surg. **8**:478, 1921. (*b*) Smithy, H. G., and Chamberlin, J. A.: Persistence of the Vitelline (Omphalomesenteric) Artery as a Clinical Problem, Surg., Gynec. & Obst. **82**:579, 1946

When the proximal and distal portions of the duct undergo obliteration but some intermediate portion fails to close, a cystic structure which is attached to the umbilicus or ileum or both by a fibrous cord [6] (fig. 2*I*) results. When the proximal and distal portions of the duct fail to obliterate but the intermediate portion closes, a diverticulum of the ileum and a separate blind intestinal pocket emptying into the

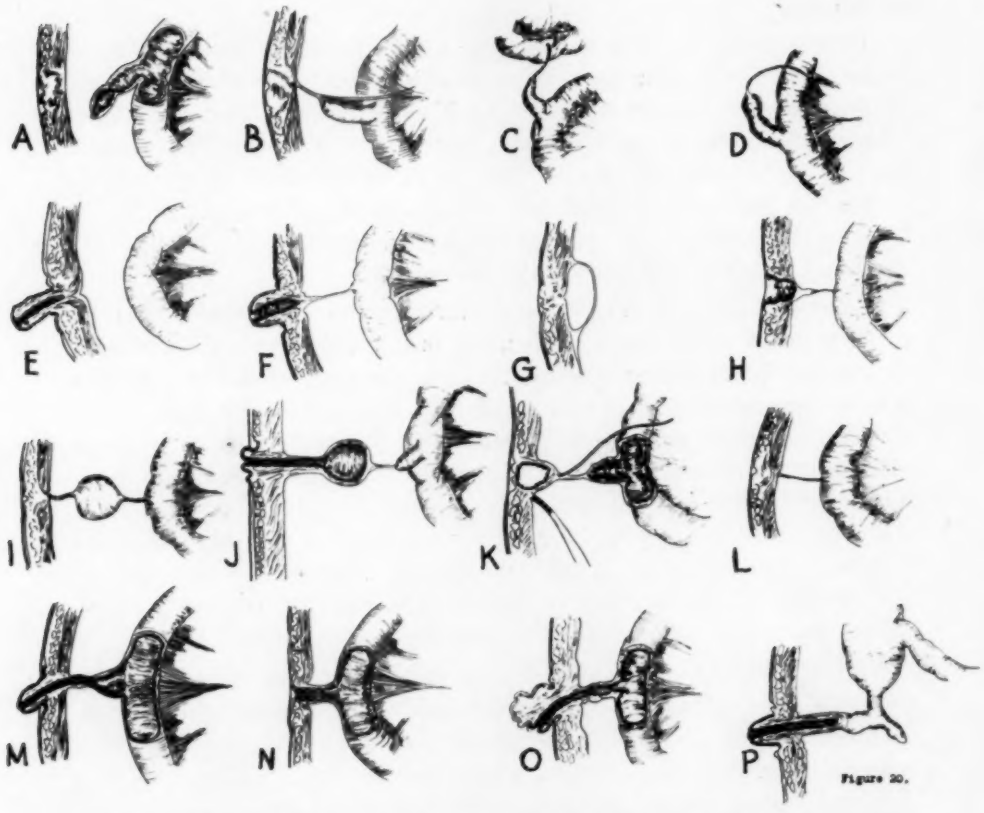

Fig. 2.—Different variations of the normal development of the omphalomesenteric duct. *A*, *B*, *C* and *D* are types in which the proximal part has failed to undergo obliteration. Distal remains of the omphalomeseneric duct are shown in *E*, *F*, *G* and *H*. *I*, *J* and *K* are forms in which varying degrees of obliteration have occurred. The remaining drawings are variations in which the omphalomesenteric duct has persisted throughout its entire course.

umbilicus are formed (fig. 2*J*). The distal portion may form a cyst of the umbilicus. There may also be remnants of the vitelline vessels

6. Cavazzini, T.: Sopra un caso di persistenza dell dotto omfalomesenterico nell'ombelico, Gazz. d. osp. **21**:472, 1900. Rupp, A.: Ein Fall von persistierenden Ductus omphaloentericus, München. med. Wchnschr. **58**:85, 1911.

or the urachus [7] (fig. 2 *K*). Residuals of the vitelline vessels or of the obliterated duct may lead to a fibrous band between the umbilicus and the ileum (fig. 2 *L*).

When no part of the omphalomesenteric duct obliterates, an external fistula of the umbilicus communicating with the ileum or an umbilical fecal fistula of Meckel's diverticulum occurs. This may form an elevated mucosal surface at the umbilicus (fig. 2*M*), or the fistula may simply open at the umbilicus (fig. 2*N*). In some instances the fistula may be associated with a polypoid growth at the umbilicus (fig. 2*O*). The duct has been described by some authors [8] as originating from the vermiform appendix (fig. 2*P*).

ANATOMY AND PATHOLOGY

Structure.—Although Meckel's diverticulum averages 5 cm. in length,[9] many giant diverticula have been reported. A diverticulum 96 cm. long has been described by Chaffin.[10] Tisdall [11] gave the case of one 59 cm. in length; he stated the belief that this represented persistence of both vitelline duct and yolk sac. Moll [12] cited the example of a diverticulum 84 cm. long. Rokitansky [13] expressed the opinion that the maximum length was 25 cm. Pollard [14] reported an intestinal diverticulum terminating at the umbilicus after a course of 90 cm.

7. (*a*) Auslander, M. M., and McClure, L.: Patent Omphalomesenteric Duct Associated with Incompletely Patent Urachus, Am. J. Dis. Child. **40**:1276 (Dec.) 1930. (*b*) Davis, H. H., and Niehaus, F. W.: Persistent Omphalomesenteric Duct and Urachus in the Same Case, J. A. M. A. **86**:685 (March 6) 1926. (*c*) Diessl, F.: Ein Fall von Dottergangfistel, Deutsche Ztschr. f. Chir. **201**:266, 1927. (*d*) Heflin, T., cited by Cullen,[1b] p. 492. (*e*) Lexer, E.: Ueber die Behandlung der Urachusfistel, Arch. f. Chir. **57**:73, 1898.

8. (*a*) Crymble, P. T.: A Case of Persistent Vitelline Duct Attached to the Vermiform Appendix, Brit. J. Surg. **9**:304, 1921; A Remarkable Vitelline Duct, Lancet **1**:1143, 1921. (*b*) Hadley, M. N., and Cogswheel, H. D.: Unusual Origin of a Meckel's Diverticulum from the Base of the Appendix, J. A. M. A. **106**:537 (Feb. 15) 1936. (*c*) Walthard, B.: Ueber die Kombination von Nabelfistel und Verdoppelung des Wurmfortsatzes, Deutsche Ztschr. f. Chir. **230**:413, 1931. (*d*) Wollmann, B.: Ueber kongenitale Nabelfisteln und Missbildungen des Ductus omphalomesentericus, Deutsche med. Wchnschr. **52**:105, 1926.

9. Jackson, C. M., and Blount, R. F.: Morris' Human Anatomy, ed. 10, Philadelphia, The Blakiston Company 1942.

10. Chaffin, L.: Surgical Emergencies During Childhood Caused by Meckel's Diverticulum. Tr. West. S. A. **49**:143, 1939.

11. Tisdall, F. F.: An Unusual Meckel's Diverticulum as a Cause of Intestinal Hemorrhage, Am. J. Dis. Child. **36**:1218 (Dec.) 1928.

12. Moll, H. H.: Giant Meckel's Diverticulum, Brit. J. Surg. **14**:176, 1926

13. von Rokitansky, L.: Lehrbuch der pathologischen Anatomie, 1861, vol. 3, p. 182; cited by Moll.[12]

14. Pollard, B.: A Diverticulum Ilei of Unusual Length and Position, Tr. Path. Soc. London **47**:47, 1896, cited by Moll.[12]

Whether many of these are intestinal reduplications or true Meckel's diverticula is questionable. Ladd and Gross [15] have presented 18 cases of duplications of the alimentary tract, a category in which some of these giant diverticula undoubtedly belong.

In form, the diverticulum of Meckel is usually cylindric, with a tapering end, it being slightly smaller in diameter than the portion of the intestine from which it originates. It may, however, have many variations. Hernia or secondary diverticula have been observed,[16] while some authors [17] have noticed bifurcation. Future pathologic change is correlated with its form, a long slender diverticulum being predisposed to intestinal obstruction, while one with a large opening at the base is more likely destined for intussusception. The size of the enterodiverticular opening is usually that of the intestine or smaller.

The usual site of Meckel's diverticulum and the one compatible with its embryology is antimesenteric, although it may be placed eccentrically,[1d] and rare cases have been reported in which it apparently appeared between leaves of the normal mesentery.[18] It is located at varying distances above the ileocecal valve, averaging 80 cm.[9] In 63 cases of Meckel's diverticulum in children, Christie [1a] observed it to occur between 15 and 90 cm. from the cecum, with an average distance of 50.6 cm. Four cases have been noticed in which the diverticulum arose from the vermiform appendix.[8] Lamb [19] stated that of 181 cases of Meckel's diverticulum it arose from the ileum in 160, the jejunum in 14 and the duodenum in the remaining few.

The diverticulum receives its blood supply either from the adjacent intestinal walls or from an independent source. Not infrequently there is a mesentery which arises from the anterior aspect of the small

15. Ladd, W. E., and Gross, R. E.: Surgical Treatment of Duplications of the Alimentary Tract: Enterogenous Cysts, Enteric Cysts, or Ileum Duplex, Surg., Gynec. & Obst. **70**:295, 1940.

16. (*a*) Curd, H. H.: A Histologic Study of Meckel's Diverticulum with Special Reference to Heterotopic Tissues, Arch. Surg. **32**:506 (March) 1936. (*b*) Fitz, R.: Persistent Omphalomesenteric Remains: Their Importance in the Causation of Intestinal Duplication, Cyst-Formation, and Obstruction, Am. J. M. Sc. **88**:30, 1884. (*c*) Hudson, H. W.: Meckel's Diverticulum in Children: Second Clinical and Pathologic Study with a Report of Thirteen Additional Cases, New England J. Med. **208**:525, 1933. (*d*) Kaspar, F.: Ein Cylindrom des Meckelschen Divertikels, Deutsche Ztschr. f. Chir. **128**:612, 1914. (*e*) King, T. W.: Feculent Discharge at the Umbilicus from Communication with the Diverticulum Ilei, Guy's Hosp. Rep. **8**:467, 1843.

17. Christie.[1a] Doepfner, K.: Beiträge zur Kenntnis des Meckelschen Divertikels, Deutsche Ztschr. f. Chir. **109**:396, 1911. Fitz.[16b]

18. Abt. I. A., and Strauss, A. A.: Meckel's Diverticulum as a Cause of Intestinal Hemorrhage, J. A. M. A. **87**:991 (Sept. 25) 1926.

19. Lamb, D. S.: The Meckel Diverticulum, Am. J. M. Sc. **105**:633, 1893.

intestinal mesentery.[20] The artery, if one is present, courses in the free border of the mesenteriole. The remnant of a vitelline vessel may persist, and this is differentiated from an obliterated diverticulum of Meckel, since the vessel always joins the mesentery and not the free, or antimesenteric, border of the intestine.[21]

HETEROTOPIA

The structure of the diverticulum mucosa is usually that of the small intestine, although heterotopic tissue was observed in 12 to 61 per cent of reported cases. Some of these studies were based on diseased diverticula, which does not represent an unselected series of specimens. However, Matt and Timpone [22] stated that in 25 per cent of all Meckel's diverticula heterotopic tissue is present.

Schaetz,[23] in thirty-seven specimens of Meckel's diverticulum, found gastric mucosa in 16.6 per cent, duodenal or jejunal mucosa in 10 per cent, pancreatic tissue in 2.7 per cent and both pancreatic and gastric tissue in 5.4 per cent. Koch [24] discovered gastric mucosa in six of the fifty specimens he examined. In 52 per cent of the twenty-three examples which Hudson and Koplik [25] studied, gastric mucosa was present. These authors also described a diverticulum containing colon mucosa.

Greenblatt, Pund and Chaney [26] reported a case in which tissue identical with mucosa of the biliary duct was present with gastric and Brunner's glands and pancreatic tissue. Though gastric tissue is commoner, pancreatic heterotopia was the first to be recognized, being described by Zenker [27] in 1861, gastric glands in Meckel's diverticulum

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20. (*a*) Christie.[1a] (*b*) Konig, P.: Zur Therapie der Dottergangfistel, Therap. Rundschau **2**:679, 1908. (*c*) Meckel, J. F.: Ueber die Divertikel am Darmkanal, Arch. f. d. Physiol. **9**:421, 1809.

21. Barrington-Ward, L.: The Abdominal Surgery of Children, London, Oxford University Press, 1937.

22. Matt, J. G., and Timpone, P. J.: Peptic Ulcer of Meckel's Diverticulum: Case Report with Review of Literature, Am. J. Surg. **47**:612, 1940.

23. Schaetz, G.: Beiträge zur Morphologie des Meckel'schen Divertikels, Beitr. z. path. Anat. u. z. allg. Path. **74**:115, 1925.

24. Koch: Missbildungen des Magens und Darmes, in Henke, F., and Lubarsch, O.: Handbuch des speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 4; cited by Neff.[3]

25. Hudson, H. W., and Koplik, L. H.: Meckel's Diverticulum in Children: Clinical and Pathologic Study, with Report of Thirty-Two Cases, New England J. Med. **206**:827, 1932.

26. Greenblatt, R. B.; Pund, E. R., and Chaney, R. H.: Meckel's Diverticulum; Analysis of Eighteen Cases with Report of One Tumor, Am. J. Surg. **31**:285, 1936.

27. Zenker, F. A.: Nebenpancreas in der Darmwand, Virchows Arch. f. path. Anat. **21**:369, 1861.

being mentioned later. Tillmans recognized gastric secretion and glands in an umbilical polyp in 1882.[28] Both pancreatic and gastric tissue may be present in the same diverticulum.

All types of gastric mucosa have been described. Both the acinar and the islet tissues of the pancreas have been described.[29] Von Rosthorn [30] found tissue in an umbilical fistula which he diagnosed as parotid. It is probable that this was pancreatic. In 1 of the cases presented in this paper, ectopic gastric and colon mucosa were present. Heterotopia is of interest because of the peptic or acid ulcers which complicate such diverticula and the relation to formation of gastric and duodenal ulcers. Dragstedt [31] showed that the diverticular gastric mucosa was capable of secreting hydrochloric acid and pepsin. These ulcers most commonly occur at the base of the diverticulum, at the junction of the gastric and ileal mucosa. Multiple ulcers have been described.[32]

A review of the theories attempting to explain the origin of these ectopic tissues was given by Greenblatt, Pund and Chaney and by Nicholson.[33] Johnston and Renner [34] have compiled 58 cases of proved peptic ulcer of Meckel's diverticulum with microscopic demonstration of gastric mucosa. They have recently [35] had another case, that of a 4 year old boy with hemorrhage, making a total of 59 in their series. Matt and Timpone [22] have collected 77 cases of histologically proved peptic ulcer of Meckel's diverticulum. Maddock and Coventry [36] added a further case to the list.

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28. Tillmans, H.: Ueber angeborenen Prolaps von Magenschleimhaus durch den Nabelring (Ectopia ventriculi) und ueber sonstige Geschwulste und Fisteln des Nabels, Deutsche Ztschr. f. Chir. **18**:161, 1882.

29. Hunt, V. C., and Bonesteel, H. T. S.: Meckel's Diverticulum Containing Aberrant Pancreas, Arch. Surg. **28**:425 (March) 1934.

30. von Rosthorn: Ein Beitrag zur Kenntniss der angeborenen Nabelfisteln, Wien. klin. Wchnschr. **2**:125, 1889; cited by Curd.[16a]

31. Dragstedt, L. R.: Ulcus Acidum of Meckel's Diverticulum, J. A. M. A. **101**:20 (July 1) 1933.

32. Peterman, M. G., and Seeger, S. J.: Meckel's Diverticulum with Hemorrhage, Am. J. Dis. Child. **36**:515 (Sept.) 1928.

33. (*a*) Greenblatt, Pund and Chaney.[26] (*b*) Nicholson, G. W.: Gastric Glands in the Extroverted Distal End of the Vitelline Duct, J. Path. & Bact. **25**:201, 1922. (*c*) Nicholson, G. W.: The Heteromorphoses in the Human Body, Guy's Hosp. Rep. **72**:75, 1922.

34. Johnston, L. B., and Renner, G., Jr.: Peptic Ulcer of Meckel's Diverticulum, Surg., Gynec. & Obst. **49**:198, 1934.

35. Johnston, L. B., and Renner, G., Jr.: Personal communication to the authors.

36. Maddock, W. G., and Coventry, M. B.: A Perforated Ulcer of the Ileum Opposite a Meckel's Diverticulum, Surg., Gynec. & Obst. **73**:105, 1941.

SEX INCIDENCE

The sex incidence of Meckel's diverticulum, as well as the complications resulting from its anomalous development, is higher in males. Curd [16a] gave the occurrence in 670 cases as 3.1 times greater in males than in females. It has been observed that in cases of pathologic changes or complications the preponderance of males is even greater. Johnston and Renner,[34] in 58 cases of peptic ulceration of Meckel's diverticulum, found 49 males and 9 females, giving a ratio of 5.4 to 1. Wellington,[37] reviewing 326 cases of diseased Meckel's diverticula, found that 201 patients were males and 64 females and that there were 61 whose sex was not stated. This is a ratio among the known cases of 3.1 males per female.

No suitable explanation has been suggested for this sexual preference. In all probability it is linked with some embryologic process during the fourth to sixth week of development, since this is the time of normal obliteration of the omphalomesenteric duct.

OCCURRENCE WITH OTHER CONGENITAL LESIONS

Frequently other malformations have been observed concomitantly with Meckel's diverticulum and its anomalous forms. Meckel,[20c] in one of his early descriptions (1809), cited its presence in patients who had such anomalies as harelip, cleft palate, bicornuate uterus, spina bifida and anencephaly. Lamb [19] added to this list its appearance in cases with visceral transposition, umbilical hernia, absent cardiac septum, atresia of the anus and extrophy of the bladder. As a companion to mongolism and to pyloric stenosis are 2 cases cited by Chaffin.[38] In one third of Christie's cases [18] of this condition occurring in childhood, malformations other than Meckel's diverticulum were present. Some of these were microgyria, microcephaly, laryngeal stenosis, esophageal atresia, ureteral anomalies and congenital cardiac conditions. In half of his cases there were umbilical hernias. He noted that 80.9 per cent of the malformations were in males and stated that "the coincidence of other anomalies . . . is offered as evidence of a defective germ plasm as an etiologic factor."

Whether remains of the vitelline stalk represent normal development, a localized ontogenetic aberration or part of a generalized disturbance of growth deserves consideration. The occurrence of Meckel's diverticulum in acceptedly normal persons is evidence against any diffuse abnormal embryology. Previous authors, however, have suggested

37. Wellington, J. R.: Meckel's Diverticulum, Surg., Gynec. & Obst. **16**:74, 1913.

38. Chaffin, L.: Surgical Emergencies during Childhood Caused by Meckel's Diverticulum, Ann. Surg. **113**:47, 1941.

that its incidence is increased when other anomalies are present. In the past ten years five thousand postmortem examinations performed at the Chicago Lying-In Hospital of the University of Chicago on fetuses and infants have revealed only 10 cases of Meckel's diverticulum. This is an incidence of 0.5 per cent, much below the normal frequency of Meckel's diverticulum. Such a low rate of occurrence in a series composed of many malformations suggests that Meckel's diverticulum is an irregularity in local growth rather than a generalized disturbance.

CLINICAL PATHOLOGY

Meckel's diverticulum has a number of complications. These have been discussed elsewhere.[39] Among the special manifestations of Meckel's diverticulum, diverticulitis has been considered by Griffith,[1c] herniation by Strohl and McArthur,[40] peptic ulcer by Matt and Timpone [22] and intussusception by Harkins.[41] Malignant growth arising within Meckel's diverticulum has been observed,[42] and even calculi have been described.[43] Various infections have been reported.[44]

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39. Barrington-Ward.[21] Christie.[1a] Christopher, F.: The Surgical Diseases of Meckel's Diverticulum, Internat. Clin. **1**:67, 1925. Cullen.[1b] Edwards, H. C.: Diverticula and Diverticulitis of the Intestine, Baltimore, William Wood & Company, 1939. Fraser and McCartney.[5a] Halstead, A. E.: Intestinal Obstruction from Meckel's Diverticulum, Ann. Surg. **35**:471, 1902, Hudson.[16c] Hudson and Koplik.[25] Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941. Miller, R. H., and Wallace, R. H.: Meckel's Diverticulum in Acute Abdominal Emergencies, Ann. Surg. **98**:713, 1933. Neff.[3] Poate, H. R. G.: Volvulus of Meckel's Diverticulum, Australia & New Zealand J. Surg. **2**:351, 1938. Smithy and Chamberlin.[5b] Womack, N. A., and Siegert, R. B.: Surgical Aspects of Lesions of Meckel's Diverticulum, Ann. Surg. **108**:221, 1938.

40. Strohl, E. L., and McArthur, S. W.: Incarcerated Meckel's Diverticulum in Femoral Hernia, Arch. Surg. **38**:783 (April) 1939.

41. Harkins, H. N.: Intussusception Due to Invaginated Meckel's Diverticulum: Report of 2 Cases with Study of 160 Cases Collected from Literature, Ann. Surg. **98**:1070, 1933.

42. Collins, D. C.; Collins, F. K., and Andrews, V. L.: Ulcerating Carcinoid Tumor of Meckel's Diverticulum, Am. J. Surg. **40**:454, 1938. Cullen.[1b] Faust, L. S., and Walters, W.: Fibrosarcoma of Meckel's Diverticulum Producing Intestinal Hemorrhage, Minnesota Med. **14**:233, 1931. Golden, T., and Stout, A. P.: Smooth Muscle Tumors of the Gastro-Intestinal Tract and Retroperitoneal Tissues, Surg., Gynec. & Obst. **73**:784, 1941. Gray, H. K., and Kernohan, J. W.: Meckel's Diverticulum Associated with Intussusception and Adenocarcinoma of Ectopic Gastric Mucosa: Report of Case, J. A. M. A. **108**:1480 (May 1) 1937. Hertzog, A. J., and Carlson, L. A.: Carcinoid of Meckel's Diverticulum: Report of Two Cases, Arch. Path. **20**:587 (Oct.) 1935. Koucky, J. D., and Beck, W. C.: Perforated Leiomyoma of Meckel's Diverticulum: Report of Case, Surgery **10**:636, 1941. Nygaard, K. K., and Walters, W.: Malignant Tumors of Meckel's Diverticulum: Report of Case of Leiomyosarcoma, Arch. Surg. **35**:1159 (Dec.) 1937.

THE PATENT OMPHALOMESENTERIC DUCT

The cases of patent omphalomesenteric duct which are available in the literature have been reviewed and summarized in two tables. Table 1 presents 104 cases of uncomplicated patent omphalomesenteric duct. Table 2 lists 27 additional cases in which this condition was complicated by a prolapse of the intestine through the umbilical fistula.

A few of the cases reported in the literature deserve special comment because of their unusual features. Crymble's case [8a] is interesting because of the origin of the duct from the appendix. Hadley and Cogswheel,[8b] Walthard [8c] and Wollmann [8d] reported similar cases except that complete patency was not present. Walthard expressed the opinion that it is embryologically untenable for Meckel's diverticulum to originate from such a site and attributed this unusual formation to the "doubling" of an unusually long appendix.

The two examples related by Garrett [45] were in twins which were believed to be homologous. In the infant of Mathews,[46] Meckel's diverticulum was noted in the umbilical hernia and was ligated in tying the cord. If the cord had sloughed away, a fecal fistula undoubtedly would have resulted. Several cases of patent omphalomesenteric duct were first recognized [1b] because of intestinal worms which escaped at the umbilicus. Recently, Mikhelson [47] described a 7 year old boy with a spontaneously discharging umbilicus from which many ascaris were obtained.

CASE 1.—A male baby was born at the Chicago Lying-In Hospital of the University of Chicago at full term, September 1943. He was delivered by low forceps and weighed 3,380 Gm. at birth. The third day of life an umbilical exudate resembling pus was noticed. This continued, and on the sixth day a 2 by 2 cm. mass of granulation tissue was observed at the navel. The discharge had ceased by this time. On the next day the umbilical protrusion showed a definite opening, from which yellow matter resembling feces and gas was expelled. Sulfathiazole locally and orally was given to control any infection present. On the eighth day a dull probe could be inserted through the umbilical aperture for 2.5 cm. The umbilical mass was most noticeable when the infant strained or cried (fig. 3*B*). The abdomen was moderately distended; tympanites was present. Sixty cubic centimeters of thin barium sulfate was injected into the umbilical fistula on the tenth day. Subsequent roentgenograms showed a communication to the small bowel, and a diagnosis of patent omphalomesenteric duct was made. It was decided to postpone operation until the infant had gained more weight and

43. Gile, J. F., and MacCarty, W. C., Jr.: Calcified Concretions Within Meckel's Diverticulum, Radiology **41**:494, 1943.

44. Cullen.[1b] Michael, P.: Tuberculosis of Meckel's Diverticulum, Arch. Surg. **25**:1152 (Dec.) 1932.

45. Garrett, J. R.: Persistent Vitelline Duct in Twins, Brit. M. J. **1**:645, 1918.

46. Mathews, F. S.: Meckel's Diverticulum, Arch. Surg. **10**:720 (March) 1925.

47. Mikhelson, A. I.: Late Umbilical Fistulas in Children, Soviet. Med. **11**:37, 1941.

TABLE 1.—*Uncomplicated Cases of Persistent Omphalomesenteric Duct*

No.	Author	Sex	Age	Treatment	Result	Comment
1	Abrahams, R.: Pediatrics **9**: 52, 1900	F	7 days	Ligation; excision	Successful	Umbilical mass 3.7 cm. long, which increased in length with baby's crying; feculent discharge; normal intestinal mucosa
2	Alsberg, A.: Deutsche med. Wchnschr. **18**: 1040, 1892	M	18 wk.	Resection	Death; peritonitis	Yellow fluid discharge; duct 4.5 cm. long
3	Anderson, W.: Tr. Path. Soc. London **42**: 128, 1891	..	Birth	None	.....	Child born at seventh month; rectum, sigmoid colon and anus absent; duct was patent
4	Ardouin, P.: Arch. prov. de chir. **17**: 1, 1908	M	3 wk.	Resection	Successful	Blood and feces discharged at navel
5	Auslander and McClure [7a]	F	2 wk.	None	.....	Fecal umbilical discharge; 1 mo. premature (birth weight 4 lb. 5 oz. [1,954 Gm.]); at autopsy: duct 15 cm. from cecum, partially patent urachus, prominent round ligament of liver
6	Auvard, A.: Travaux d'obstetrique, Paris, Lecrosnier et Babé, 1889, Book I, p. 331	M	Birth	None	.....	Child died on thirty-fourth day; meconium and thick red-green fluid discharged at navel; duct 8 cm. long, 42 cm. from cecum
7.	Battle, W. H.: Tr. Clin. Soc. London **26**: 237, 1893	F	18 mo.	Resection	Successful, but death from scarlet fever in 11 days	Fecal discharge; duct 10 in. from ileocecal valve
8	Baum, cited by Morian: Arch. f. klin. chir. **58**: 306, 1899	M	4 mo.	Bandaged	Successful	
9	Billroth, C. A. T.: Chirurgische Klinik, Berlin, A. Hirschwald, 1869, p. 294	M	14 wk.	Ligation	Death	Feculent discharge from duct
10	Briddon, C. K.: Ann. Surg. **28**: 647, 1898	M	Birth	Reduction; resection	Successful	Feces from umbilical opening; duct 12 in. from ileocecal valve; operation done in 2 stages
11	Brindeau: Nouv. Arch. d'obst. et de gynec. **25**: 45, 1895	..	5 days	None	.....	Patient died on fifth day of life; duct 4.5 inches above cecum; patient weighed 2 lb. 3 oz. (991 Gm.); direct connection between lumen of bowel and liquor amnii before birth
12	Broadbent: M. Times & Gaz. **2**: 45, 1866	..	.....	None	.....	Autopsy specimen: fecal umbilical fistula and congenital syphilis; intestine in contact with abdominal wall
13	Broca, A.: Rev. d'orthop. **6**: 47, 1895	M	6 mo.	Resection	Successful	Mucous discharge at umbilicus
14	Broca, J., cited by Bureau: Thesis, Paris, no. 257, 1898, p. 32	M	10 mo.	Resection	Successful	Serous discharge from fistula
15	Brun, L. A.: Thesis, Paris, no. 238, 1834	..	28 days	Ligation	Successful	Fecal discharge (case reported by Dupuytren)
16	Brun, L. A.: Thesis, Paris, no. 238, 1834	M	9 days	Ligation	Successful	Fecal discharge (case reported by Dupuytren)
17	Caldbick, S. L.: S. Clin. North America **8**: 1341, 1928	M	5 days	Resection	Successful	Fecal discharge; duct 3.7 cm. long
18	Cheyne, W.: Brit. M. J. **1**: 815, 1892	..	3 wk.	None	.....	Imperforate rectum, anus patent for 1 in.; fecal discharge
19	Clarkson, G. A.: Brit. M. J. **1**: 700, 1901	..	Infant	.....	.....	Umbilical fecal discharge
20	Crymble [8a]	M	5 mo.	Resection	.....	8 cm. diverticulum attached to appendix; intestinal

19	Clarkson, G. A.: Brit. M. J. **1**: 700, 1901	..	Infant	.....	.....	Umbilical fecal discharge
20	Crymble [8a]	M	5 mo.	Resection	.....	8 cm. diverticulum attached to appendix; intestinal mucosa
21	Czerey-Pechany, von A.: Zentralbl. f. Chir. **69**: 795, 1942	M	18 mo.	Resection	Successful	Umbilical skin excoriated
22	DeLucia, A. E.: Ann di med. nav. e colon. **2**: 315, 1916	M	3 yr.	Resection	Successful	Feces discharged at umbilicus
23	Deschin: Zentralbl. f. Chir. **22**: 1154, 1895	M	5 mo.	Resection	Death	Fecal discharge: duct 48 cm. above cecum; mucosa was intestinal type
24	Eid, F. L.: Canad. M. A. J. **23**: 676, 1930	M	12 mo.	Resection	.....	Mucous secretion; duct 8.8 cm. long
25	Eves, A.: Lancet **1**: 101, 1845	..	1 mo.	Ligation	Successful	Fecal discharge
26	Fitz [16b]	M	21 yr.	None	.....	Patient died later from intestinal obstruction; intermittent fecal umbilical discharge during lifetime; duct 120 cm. above cecum
27	Forster, cited by King [16c]	M	14 mo.	Excision, locally	Successful	Serofeculent discharge; probe admitted for 5 cm.
28	Froelich, R.: Rev. mens. d. mal. de l'enf. **20**: 517, 1902	M	5 yr.	Resection	.....	Serosanguineous umbilical discharge; intestinal mucosa
29	Gampert: Rev. méd. de la Suisse Rom. **13**: 356, 1893	M	5 days	Ligation	Successful	Fecal discharge; mass of size of a "little finger" at umbilicus
30	Garrett [45]	F	10 days	None	.....	Patients were twins "likely to be homologous twins"; bile discharge in small quantities at umbilicus
32	Gevaert, G.: Ann. de méd. **4**: 1, 1892	M	30 mo.	Resection	Successful	Fecal discharge at umbilicus
33	Goodhart, G. W.: Tr. M. Soc., London **62**: 107, 1939	..	.....	.....	.....	Specimen described with patency to exterior; lined by intestinal mucosa; serous discharge; no feces
34	Hansen, J. A.: Ein Beitrag zir Persistenz des Ductus omphalo-entericus, Inaug. Dissert., Kiel, Lipsius & Tischer, 1885	..	8 days	Ligation	Successful	Fecal contents discharged; slight bleeding at umbilicus
35	Heaton, G.: Brit. M. J. **1**: 627, 1898	M	4 days	Reduction	Death 2 wk. later	Umbilical fecal discharge until all feces passed by this aperture; duct 17.5 cm. from cecum; ascending colon left side of spine, hepatic flexure in normal position; duct lined with intestinal mucosa
36	Heflin [48]	..	14 mo.	Resection	Successful	Bleeding from umbilicus which began 2 to 3 days postnatally and continued until child became anemic; umbilical discharge like urine and feces; operative diagnosis of patent urachus and omphalomesenteric duct
37	Hickman: Tr. Path. Soc., London **20**: 418, 1869	F	4 mo.	None	.....	Fecal odor to discharge
38	Holmes, T.: Surgical Treatment of Diseases of Infancy and Childhood, London, 1868, p. 182	M	10 yr.	None	.....	Intestinal contents discharged
39	Hubbard, J. C.: Ann. Surg. **35**: 495, 1902	F	1 mo.	Resection	Successful	Red bleeding mass at umbilicus
40	Hudson [16c]	M	3 wk.	Resection	Successful	Intestinal type of mucosa; child also had inguinal hernia, harelip, cleft palate and total congenital absence of eyes
41	Jacoby, M.: Berl. klin. Wchnschr **14**: 202, 1877	M	6 days	Reduction	Successful	Fecal umbilical discharge
42	Kehr, H.: Deutsche med. Wchnschr. **18**: 1166, 1892	M	28 yr.	Resection	Successful	Mucous discharge
43	Kern: Beitr. z. klin. Chir. **19**: 353, 1897	M	6 mo.	Resection	Successful	Green fluid discharge

TABLE 1.—*Uncomplicated Cases of Persistent Omphalomesenteric Duct—Continued*

No.	Author	Sex	Age	Treatment	Result	Comment
44	King [16e]	M	8 days	None	.....	Child died from "thoracic disease"; duct 12.5 cm. long, 45 cm. from cecum
45	Kirmisson, E.: Rev. d'orthop. **2**: 321, 1901	M	22 wk.	Resection	Successful	Strawberry-sized red umbilical mass
46	Konig [20b]	M	1 to 2 wk.	Resection	Successful	Mucous exudate; patency demonstrated at operation
47	Korte: Deutsche med. Wchnschr. **24**: 103, 1898	M	15 mo.	Resection	Successful	Duct originated from mesenteric side of intestine
48	Lamare, J. P.; Courtois, J., and Isidore, P.: Gynécologie **37**: 193, 1938	M	2 mo.	Resection	Successful	Diagnosed by injecting iodized poppyseed oil; intestinal mucosa
49	Leveuf, J.; Leroux, R., and Perrot, A.: Ann. d'anat. path. **12**: 1015, 1935	M	.....	.....	.....	3 cases presented, all patients were males; 2 had fecal umbilical discharges; ages: 4 mo., 1 yr. and 8 yr.; all had resections and recovered successfully; 1 had gastric mucosa
52	Leisrink and Alsberg: Arch. f. klin. Chir. **28**: 768, 1882	M	3 yr.	Resection	Death; peritonitis	First seen at age of 3 yr.; not operated on until 10 yr.; duct had constricted intestine, causing gangrene
53	Lexer [7e]	M	18 mo.	Resection	Successful	Purulent and a clear fluid discharged at umbilicus; symptoms relieved by correcting severe phimosis; laparotomy showed patent duct; operative diagnosis of patent urachus and vitelline duct; specimen showed intestinal mucosa with Lieberkühn's glands
54	MacSwiney, S. M.: Proc. Path. Soc., Dublin **4**: 251, 1873-1875	M	7 yr.	.....	.....	Ascaris extracted from umbilical opening; yellow discharge
55	Marshall: M. Times & Gaz. **2**: 640, 1868	M	.....	Excision externally	.....	Fecal contents discharged from umbilicus
56	Mathews [46]	M	20 hr.	Resection	Death	Small umbilical hernia with duct 6 cm. long
57	Montgomery, J. G.; Gilkey, H. M.; Kyger, F. B., and Jennings, W. L.: J. Missouri M. A. **35**: 244, 1938	M	Birth	Resection	Successful	Large umbilical hernia concomitant; meconium issued with pressure on umbilicus; duct 2 cm. long; intestinal mucosa
58	Morgan, J. E.: Am. J. Surg. **58**: 267, 1942	M	Birth	Resection	Successful	Feces and gas expelled at umbilical orifice; artery along duct; ileal mucosa, no heterotopia
59	Morian: Arch. f. klin. Chir. **58**: 306, 1899	M	4 days	Resection	Successful	Umbilical fecal discharge; operation at 5 wk.
60	Muresanu, E.: Cluj. med. **9**: 158, 1928	M	8½ yr.	Resection	Successful	Congenital serous discharge; umbilical area traumatized at age 8½ yr., with much bleeding subsequently
61	Neurath, R.: Wien. klin. Wchnschr. **9**: 1158, 1896	..	13 days	Resection	Successful	6 cm. long duct; intestinal mucosa
62	Nicaise, in Dictionnaire encyclopédique des sciences médicales, Paris, 1881, vol. 15, p. 159	..	Birth	Cauterization	Successful	Fecal discharge; case observed by Patry
63	Nicholson [33b]	F	5 mo.	Ligation, externally	Successful	Gastric mucosa at umbilicus; intestinal contents discharged
64	Nicolas, P.: Sur deux variétés de fistules ombilicales, Paris, A. Parent, 1883	M	6 days	None	Death	Purulent umbilical discharge and fecal emesis at birth; obstruction in rectum; small bowel 2½ times normal diameter; 3 cm. long duct; imperforate sigmoid
65	O'Neil, W. E.: S. Clin. **3**: 541, 1919	M	38 wk.	Resection	Successful	Tongue tied, phimosis, supernumerary finger; blood oozed at umbilicus

65	O'Neil, W. E.: S. Clin. **3**: 541, 1919.....	M	38 wk.	Resection	Successful	Tongue tied, phimosis, supernumerary finger; blood oozed at umbilicus; patency shown by roentgenogram
66	Orth, J.: Lehrbuch der speciellen pathologischen Anatomie, Berlin, A. Hirschwald, 1887, vol. 1, p. 765	..	Birth	.....	.....	"I recently made an autopsy on a new-born child and found a diverticulum split longitudinally below the umbilicus and adherent to the anterior abdominal wall in such a manner that meconium could escape into the abdominal cavity. A large quantity of meconium lay between the abdominal wall and omentum"
67	Ott, I.: Riforma med. **45**: 615, 1929.....	M	4 mo.	Resection	Successful	Feces and gas expelled at umbilicus
68	Park, R.: Med. Fortnightly **9**: 9, 1896.....	M	Adult	Resection	Successful	Always a "slight discharge from the navel"
69	Parker, C. H.: Am. J. Roentgenol. **10**: 605, 1923.....	..	3 wk.	None	.....	Fecal discharge at umbilicus
70	Pautiennis, K.: Medicina, Kaunas **18**: 1002, 1937.....	..	3 yr.	Resection	Successful	Mucous discharge at umbilicus; duct was 7 cm. long
71	Pernice, L.: Die Nabelgeschwulste, Halle, Max Niemeyer, 1892.	M	7 mo.	Curettage, externally	Successful	Mucus and particles of feces at umbilicus, especially when child cried; umbilical skin excoriated
72	de Planque, P. M.: Nederl. tijdschr. v. geneesk. **1**: 3177, 1928	M	2 yr.	Resection	Successful	Umbilical hernia since birth; fecal discharge for about 6 mo. preoperatively
73	Poussin: J. de méd., chir., pharm., Paris **40**: 81, 1817	M	3 yr.	None	.....	Nurse pulled cord away on fifth day; subsequent fecal discharge; Ascaris through umbilical opening
74	Pratt, J. W.: Lancet **2**: 1142, 1884.....	M	33 wk.	Ligation, externally	Successful	Fecal umbilical discharge, occasionally hemorrhage
75	Prestat, cited by Ledderhose, G., in Die chirurgischen Erkrankungen der Bauchdecken und die chirurgischen Krankheit der Milz. in Deutsche Chirurgie, Stuttgart, F. Enke, 1890, no. 45b	M	Birth	None	.....	No fistula, but on opening abdomen (reason not stated), a duct was found extending from the ileum to the abdominal wall; pressure on small bowel caused rupture of umbilical cicatrix, fecal discharge to the exterior
76	Quaet, G.: Das Offenbleiben des Ductus omphalomesentericus, Kiel, Peters, 1899	M	9 days	Resection, externally	Successful	Fecal discharge
77	Quaet, G.: Das Offenbleiben des Ductus omphalomesentericus, Kiel, Peters, 1899	M	10 mo.	Resection, externally	Successful	Mucous discharge
78	Quaet, G.: Das Offenbleiben des Ductus omphalomesentericus, Kiel, Peters, 1899	M	2 days	Resection, externally	Successful	Fecal navel discharge
79	Quaet, G.: Das Offenbleiben des Ductus omphalomesentericus, Kiel, Peters, 1899	F	5 yr.	Resection	Successful	Fluid secretion from navel
80	Railton, T. C.: Brit. M. J. **1**: 795, 1893.....	F	11 mo.	Resection, externally	Successful	Fecal discharge for several months after birth; recently only mucous; intestinal mucosa
81	Ratnayeke, N.: Brit. J. Surg. **24**: 402, 1936.....	M	6 wk.	Resection	Successful	Feculent discharge; duct 7.5 cm. long
82	Rentschler, C. B.: Arch. Surg. **40**: 694, 1940.....	M	3 mo.	Resection	Successful	Green-brown fluid discharge smelling like feces; duct 5 cm. long; intestinal mucosa
83	Rice, G. E.: Colorado Med. **24**: 14, 1927.....	F	"Years"	Bandaged	Successful	Intermittent fecal discharge which responded successfully to bandaging; operation refused
84	Robinson, H. B.: Lancet **1**: 302, 1902.....	M	"Months"	Resection	Successful	Fecal discharge; duct 4.3 cm. long

TABLE 1.—*Uncomplicated Cases of Persistent Omphalomesenteric Duct—Continued*

No.	Author	Sex	Age	Treatment	Result	Comment
85	Roth, M.: Virchows Arch. f. path. Anat. **86**: 371, 1881	M	1 mo.	None	Death at 6 mo.	Duct located 58 cm. above cecum; fecal discharge; at junction of duct with small intestine mucosa projected inward, forming a valve
86	Ryadnov, S. M.: Sovet. klin. **20**: 143, 1934.....	M	17 yr.	Resection	Successful	Fecal discharge since birth; duct 18 cm. long; functioning artery along duct
87	Salzer, H.: Wien. klin. Wchnschr. **17**: 614, 1904.....	F	5 mo.	Resection	Successful	Mucous discharge; duct 2 cm. long; externally, gastric mucosa
88	Sauer, F.: Deutsche Ztschr. f. Chir. **44**: 316, 1897...	M	9 mo.	Resection	Death; peritonitis	Duct 2.5 cm. long, arising 53 cm. above cecum; intestinal mucosa
89	Schroeder, G.: Ueber die Divertikel-Bildungen am Darm-Kanale, Inaug. Dissert., Augsburg, Volkhart, 1854	..	6 mo.	None	.....	This is a specimen in the Prague pathologic museum
90	Semb, O.: Norsk mag. f. lægevidensk. **83**: 778, 1922...	M	3½ wk.	Resection	Successful	Large umbilical hernia present
91	Shepherd, F. J.: Arch. Pediat. **9**: 55, 1892.....	M	3 mo.	Resection	Successful	Fecal discharge; functional omphalomesenteric artery present; duct 6 cm. long
92	Stierlin, R.: Deutsche med. Wchnschr. **23**: 188, 1897..	M	2 mo.	Resection	Successful	Fecal discharge
93	Strasser, A. A.: M. Rec. **64**: 933, 1903.....	M	7 yr.	None	.....	Reduction helped for 1 mo.; feces at umbilicus increased with crying of child
94	Strater, M.: Deutsche Ztschr. f. Chir. **74**: 143, 1904...	M	14 days	Resection	Successful	Duct 3 to 4 cm. long; mucus discharged
95	Veit, K. E.: Arch. f. klin. Chir. **102**: 1014, 1913.....	..	10 days	Resection	Successful	Fecal discharge; mesenteriole attached to diverticulum; intestinal mucosa
96	Weiss, E.: Ueber diverticuläre Nabelhernien und die aus ihnen hervorgehenden Nabelfisteln, Inaug. Dissert., Giessen, Brühl, 1868	M	3 wk.	Caustic applications	Successful	Fecal discharge at navel
97	Weiss, E.: Ueber diverticuläre Nabelhernien und die aus ihnen hervorgehenden Nabelfisteln, Inaug. Dissert., Giessen, Brühl, 1868	M	11 wk.	Cauterization	Death at 1 yr.	Duct accompanied with mesentery; intestinal mucosa inflamed
98	Wellington [37]	M	7 yr.	Resection	Successful	Duct 2.5 cm. long; mucous discharge
99	Wilks, S.: Tr. Path. Soc., London **16**: 126, 1865.....	..	Child	"Plastic operation"	Successful	Death occurred later from intestinal obstruction
100	Wu, Y. K., and Yu, S. T.: China M. J. **60**: 264, 1941..	M	20 mo.	Resection	Successful	Mucous discharge; duct attached 45 cm. from cecum
101	Wu, Y. K., and Yu, S. T.: China M. J. **60**: 264, 1941..	M	1 mo.	None	.....	Fecal umbilical discharge
102	Wu, Y. K., and Yu, S. T.: China M. J. **60**: 264, 1941..	M	6 mo.	Resection	Successful	Fecal discharge; duct 1.5 cm long, 30 cm. from cecum; intestinal mucosa with duodenal glands
103	Morgan: Personal communication to the authors....	M	6 days	Resection	Successful	Feces discharged at umbilicus; duct 3.5 cm. long; umbilical mass became tense and larger with straining of child
104	Kittle, Jenkins and Dragstedt.....	F	5 days	Resection	Successful	Gastrocolonic mucosa; fibrous remnants of vessels accompanying duct

was stronger, in the hope that the advantage of a better general condition for operation would outweigh the hazard of a possible prolapse.

The infant was discharged but was readmitted two days later because a small portion of the omphalomesenteric duct had prolapsed. This was easily reduced manually. Little gas or fecal material was being discharged at this time. Several days later the baby was sent home.

One month later the baby was again admitted, because of vomiting of six hours' duration, restlessness and the protrusion of dark red bowel through the navel opening. About five hours before vomiting, the mother noticed that the umbilical mass was a dark red instead of its usual pink. Physical examination on admission showed a 15 cm., dull red, T-shaped loop of bowel protruding at the umbilicus. The diagnosis was made of intussusception of the ileum through the patent omphalomesenteric duct. The infant was in poor general condition. Infusion

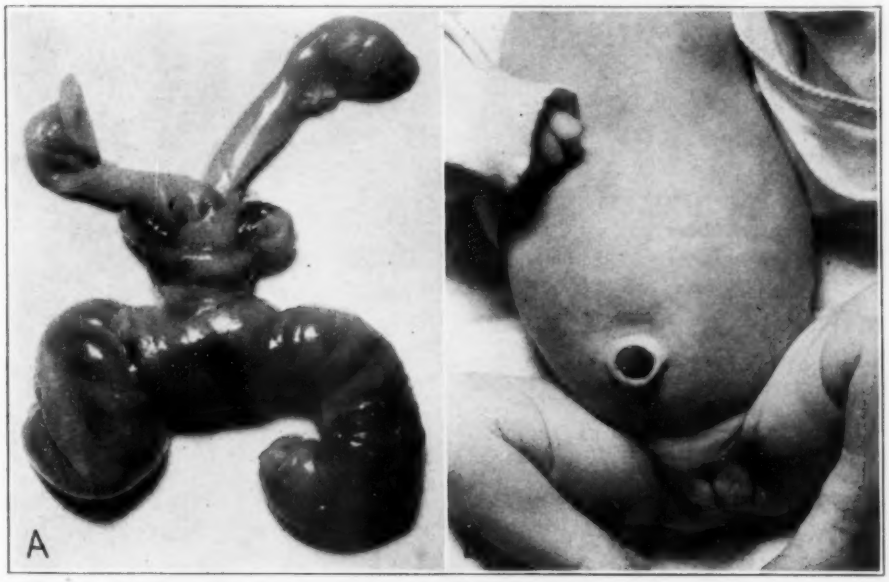


Fig. 3.—*A*, pathologic specimen removed from patient in case 1. The gangrenous T-shaped piece of prolapsed ileum is seen in the lower half of the picture. Above are the afferent and efferent limbs of normal intestine. *B*, the baby in case 2. The red mass of intestine is visible at the umbilicus. This protruded decidedly when the child cried or strained at stool.

of isotonic solution of sodium chloride was started and transfusion begun. Immediate operation was performed by Dr. L. R. Dragstedt. An elliptic incision was made about the umbilicus down to the fascia, the umbilicus was excised and the mass of prolapsed intestine was elevated. The afferent and efferent loops of ileum near the point of intussusception were transected and the stumps invaginated with purse string sutures. The mass of prolapsed intestine and the umbilicus were then removed and a side to side anastomosis performed between the two segments of ileum. The abdomen was closed with silk sutures.

The convalescence following operation was relatively smooth except for a slight febrile reaction for the first two days. On the thirteenth day, however, there was evidence of a moderately severe infection in the wound, from which beta hemolytic

streptococci were isolated. The infection responded to drainage and local treatment with sulfathiazole. The infant was discharged from the hospital six and a half weeks after operation ,in relatively good general condition except for some drainage from the umbilical incision. This was followed in the outpatient department, and the wound subsequently healed well.

The pathologic specimen (fig. 3*A*) consisted of the umbilicus, a small portion of surrounding skin and the mass of gangrenous ileum which had prolapsed through the patent omphalomesenteric duct. Microscopic sections of the bowel showed extensive hemorrhage into the mucosa and beginning necrosis. There was no evidence of aberrant tissue in the short segment of patent duct.

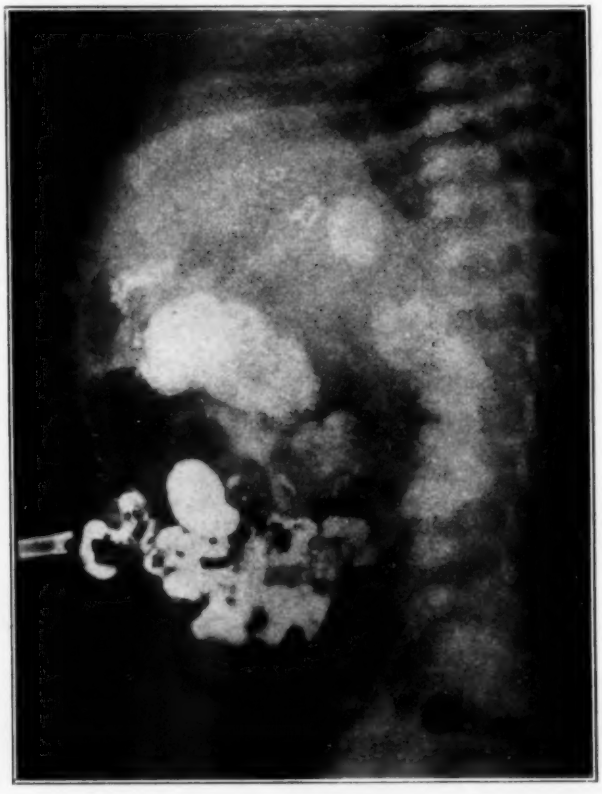


Fig. 4.—Roentgenologic demonstration of the patent omphalomesenteric duct in case 2 following the introduction of iodized oil through a catheter at the umbilical aperture. A similar roentgenogram was obtained in case 1.

CASE 2.—A female infant weighing 3,730 Gm. was born at the Chicago Lying-In Hospital of the University of Chicago in November 1944, after an uncomplicated spontaneous delivery. The only abnormality noted at birth was a large and bulbous umbilical cord. On the fifth day the umbilical stump was red and swollen, measured 3 cm. in width and protruded for a distance of 1 cm. There was a moderate amount of discharge. Because of the redness and discharge it was thought that this represented some infection of the navel, and sulfathiazole oint-

Fig. 5.—Gross specimen removed from patient in case 2. The umbilical skin and the contiguous mass of intestine are seen, with a probe passing through the lumen of the omphalomesenteric duct.

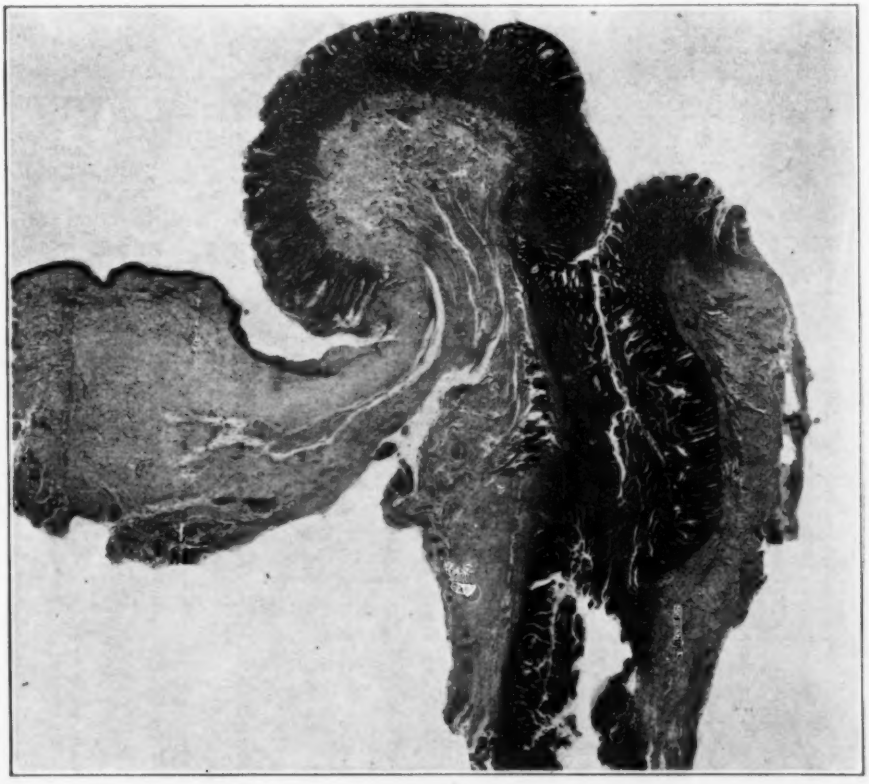


Fig. 6.—Photomicrograph of the specimen in case 2. Note the umbilical polypoid mass of intestinal mucosa, the mucocutaneous junction and the lumen of the omphalomesenteric duct.

TABLE 2.—*Patent Omphalomesenteric Duct with Prolapse of Ileum*

No.	Author	Sex	Age	Treatment	Result	Comment
1	Arndt, C.: Arch. f. Gynäk. **52**: 71, 1896.....	M	16 yr.	Reduction; resection	Death; peritonitis	Mucous and fecal discharge from umbilicus; 18 cm. of bowel prolapsed
2	Barth, A.: Deutsche Ztschr. f. Chir. **26**: 193, 1887....	M	9 days	Reduction; resection	Death; peritonitis	Umbilical fecal fistula; diagnosed on ninth day; prolapse occurred on fourteenth day
3	Basevi, S.: Jahrb. f. Kinderh. **12**: 275, 1878.....	..	12 days	None	Death	4 cm. cone projecting through umbilicus without any opening; this increased in size when child cried; umbilical raw area since cord detached; no fecal discharge; appeared on nineteenth day; 2 days later became necrotic and child died; autopsy diagnosis
4	Blin: Compt. rend. Soc. de biol. **4**: 131, 1853.....	..	6 mo.	Reduction	Death 2 days after prolapse	Fecal matter had escaped early in life; 3 to 4 cm. diverticulum 25 cm. above cecum at autopsy
5	Clamann: Deutsche med. Wchnschr. **28**: 416, 1902....	M	3½ wk.	None	Death	Umbilical fecal discharge
6	Cooper, P. R.: Clin. J. **47**: 78, 1918.....	M	7 mo.	Resection	Death	Umbilical fecal discharge; patent duct diagnosed at age of 1 wk.; prolapse 7 mo. later
7	Cserey-Pechany, von A.: Zentralbl. f. Chir. **69**: 795, 1942	F	5 wk.	Resection	Death	Mucofeculent discharge from umbilicus; patient seen after prolapse had been present for 4 days
8	Cutler, G. D: Boston M. & S. J. **190**: 782, 1924.....	..	Birth	Resection	Successful	Prolapse occurred through side of umbilical cord; condition noted at birth with immediate operation
9	Gesenius: J. f. Kinderkr. **30**: 56, 1858.....	M	25 days	None	Death	Umbilical polyp with yellow fluid discharge; 7.5 cm. prolapse on twenty-third day; duct 1 cm. long, 13.5 cm. from cecum found at autopsy
10	Gevaert, G., in Charon, E., and Gevaert, G.: Chirurgie infantile, ed. 2, Brussels, 1895, p. 251	..	6 wk.	Reduction	Death	Umbilical fecal fistula; 12 cm. prolapse
11	Golding-Bird, C. H.: Clin. Soc. Tr. **29**: 32, 1896.....	M	4 wk.	None	Death	Fecal umbilical discharge from time of cord detachment, progressing until all feces came through umbilicus at time of admission; ileum distal to duct origin prolapsed
12	Guthrie, L. G.: Pediatrics **2**: 1, 1896.....	M	3 days	None	Death	This child passed all its feces from birth through the omphalomesenteric duct; prolapse of 15 cm. at age of 6½ wk.; duct was 30 cm. above cecum; ileum greatly dilated; large bowel collapsed
13	Helweg, K.: Hospitalstid. **2**: 705, 1884.....	M	4 mo.	Reduction	Death	No umbilical mass at birth; at admission a small mass was noted; this after ligation became necrotic; shortly after, child coughed violently, and 20 cm of ileum prolapsed; duct was 45 cm. above ileocecal valve
14	Holmes. T.: Surgical Treatment of Diseases of Infancy and Childhood, London, 1868, p. 182	M	2 wk.	None	.....	For 8 days child had an umbilical mass, then a 9 cm. prolapse, which resembled a "stuffed sausage"
15	Hue, F.: Normandie méd. **21**: 162, 1903.....	M	12 days	Reduction	Successful	This boy had been born [illegible]

15	Hue, F.: Normandie méd. **21**: 162, 1903.....	M	12 days	Reduction	Successful for several days; prolapse recurred; child died	This boy had been born prematurely, with a bifurcated cord; after 2 wk. feces emerged at umbilicus; soon intestine prolapsed
16	Huttenbrenner, A.: Allg. Wien. med. Ztg. **23**: 225, 1878	..	5 mo.	None	Death	10 cm. prolapse; autopsy showed persistent left omphalomesenteric artery
17	Kolbing, A.: Neue Ztschr. f. Geburtsk. **14**: 443, 1843..	M	19 wk.	Resection	Death	Early history not given; after an attack of pertussis, 23 cm. prolapse of ileum
18	Lowenstein, L.: Arch. f. klin. Chir. **44**: 541, 1894-1895	M	3 mo	"Operation"	Death	Reddish umbilical tumor which was removed early in life; prolapse at age of 19 wk.
19	Most, A.: Beitr. z. klin. Chir. **144**: 236, 1928.....	M	Birth	.....	.....	Umbilical hernia with intestinal prolapse through omphalomesenteric duct at birth
20	Ophuls, W.: Beiträge zur Kenntnis der Divertikelbildungen am Darm-Kanale, Inaug. Dissert., Gottingen, 1895	M	3 wk.	Resection	Death; peritonitis	10 to 15 cm. of bowel had prolapsed; early history not given; duct 35 cm. above cecum
21	Siebold, quoted by Schroder, G.: Ueber die Divertikel-Bildungen am Darm-Kanale, Inaug. Dissert., Augsburg, 1854	..	3 wk.	Reduction	Death	Patent duct diagnosed at birth by mucous umbilical discharge; gangrenous area noted at third week; prolapse with crying, which increased with crying; duct (measured at autopsy) was 2 cm. long
22	Theremin: Rev. mens. d. mal. de l'enf., 1885, p. 558....	M	11 days	Reduction	Death	Umbilical tumor since cord detachment; superficial umbilical hemorrhage on twenty-third day; prolapse of 10 to 12 cm. then; autopsy confirmed diagnosis of prolapse through patent omphalomesenteric duct
23	Theremin: Rev. mens. d. mal. de l'enf., 1885, p. 558...	..	.....	Reduction	Death from "gastro-intestinal catarrh" when 2 mo. old	Umbilical mass after cord sloughing; no sign of an opening, however, some umbilical bleeding; intestinal prolapse at 1 mo.; prolapse occurred when child cried; portion prolapsed 10 cm.; prolapse receded slightly; autopsy showed complete reduction of prolapse; duct 60 cm. above cecum; bilateral posterior atelectasis
24	Veit, K. E.: Arch. f. klin. Chir. **102**: 1014, 1913.....	M	11 days	None	Death	Fecal umbilical discharge noted soon after birth; at 1 mo. a "large" prolapse occurred
25	Violbing, cited by Bureau, J.: Thesis, Paris, no. 257, 1898, p. 32	M	19 wk.	None	Death	Umbilical mass noticed after cord detached; this was tied close to base; at 19 wk. 45 cm. intestinal prolapse; autopsy confirmed diagnosis
26	Weinlechner: Jahrb. f. Kinderh. **8**: 55 1874-1875.....	M	5 mo.	Resection	Death	Umbilicus had been open since birth, with mucus, but no feces discharged; during an attack of coughing the day before admission 2 cm. prolapse had appeared; autopsy confirmed diagnosis, showed duct to originate 7.5 cm from cecum
27	Kittle, Jenkins and Dragstedt.....	M	8 days	Resection	Successful	Prolapse at age of 2 mo.; fecal umbilical discharge

ment was applied. The discharge continued, however, and on the fourteenth day a dark red, mushroom-shaped mass was noted at the umbilicus. This measured 2.0 cm. in width and 0.4 cm. in height. The surrounding skin continued to be red. In the center of this mass a small lumen was observed, which would admit a probe for a short distance.

When the infant cried the red mass noticeably protruded (fig. 3*B*). Introduction of a small amount of iodized oil into the lumen of this red umbilical mass, followed by roentgenologic examination, revealed a communication into the small intestine near the cecum (fig. 4). The diagnosis of patent omphalomesenteric duct was established and operation advised.

The infant, under a combination of local and ether anesthesia, was operated on on her thirty-third day by Dr. H. P. Jenkins. An elliptic incision down to the fascia was made close to the umbilicus. The fascia was separated from the structures of the navel and the peritoneum opened. A short segment of bowel was found to extend from the umbilicus to the ileum. This was transected at the junction with the ileum and the defect in the ileum closed over with a continuous suture of 4-0 chromic, which was reenforced with interrupted sutures of 6-0 silk. Palpation after closure revealed no impairment of the lumen of the ileum. There was an additional cordlike structure which coursed from the umbilicus to some point within the abdomen. This was fibrous in nature and when cut across revealed what appeared to be two obliterated blood vessels, probably remnants of the omphalomesenteric vessels. It was necessary to transect and ligate this fibrous cord to permit removal of the umbilicus and the patent duct. The wound was closed with interrupted 4-0 chromic surgical gut, the fascia at the umbilicus being imbricated for a more solid closure of the abdominal wall. An attempt was made to close the skin in such a way that the appearance of a normal navel would be simulated.

The infant had an uneventful convalescence. The wound healed without difficulty, and the baby was discharged in good condition two weeks after operation. When last seen, one year after operation, the child was in excellent general condition, with no evidence of herniation or umbilical discharge.

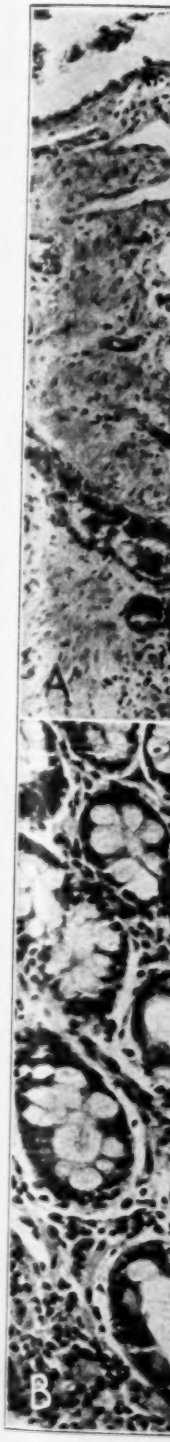
The specimen removed at operation consisted of the umbilicus, the short stump of patent duct which protruded through the umbilicus and the fibrous cordlike structure containing the obliterated vessels (fig. 5). Microscopically, gastric mucosa as well as an area of colonic mucosa (figs. 6, 7 and 8) was present.

COMMENT

In table 1, giving the uncomplicated cases, 74 patients were males and 17 were females, and in 13 cases the sex was not stated. This is a ratio of 4.4 males per female. In table 2, listing the cases in which patency was complicated by prolapse, of the 27 cases, in 19 the patients were males, only 1 patient was a female, and the sex of the others was not given. These figures definitely indicate a preponderance of males in cases of patent omphalomesenteric duct, and, although the series is limited, a certain predilection for complication in the male sex is noted.

There are 19 cases in which other anomalies have been mentioned, 5 being cases of umbilical hernia. Auslander and McClure,[7a] Heflin [48]

48. Heflin, H. T., cited by Cullen,[1b] p. 492.



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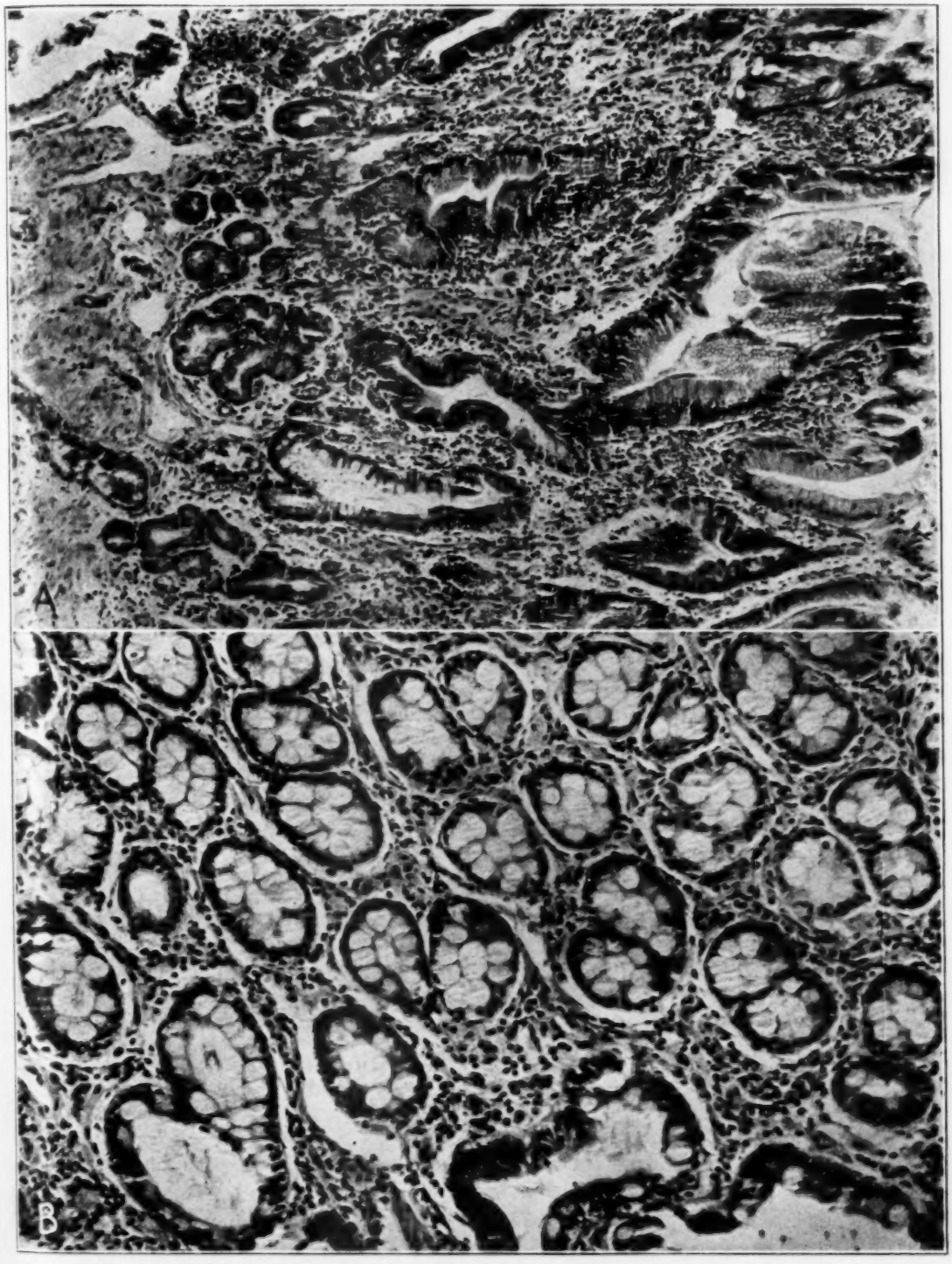

Fig. 7.—*A*, photomicrograph of the specimen removed in case 2, illustrating the mixture of gastric and colon mucosa. The deep mucosal-lined crypts are seen. *B*, the heterotopic mucosa seen at higher power.

and Lexer [7e] present patients in which urachal remnants are prominent. In 5 instances parts of omphalomesenteric vessels were recognized. Four patients showed other gastrointestinal anomalies.

In 22 cases microscopic analysis of tissue was reported. Of these, in 18 it was stated to be "intestinal mucosa" and in 4 gastric mucosa. This gives an incidence of about 18 per cent for heterotopia in the cases of patent omphalomesenteric duct in which the histology is mentioned.

No compilation of mortality statistics is of value. Patients in the older cases were moribund because of the type of operation at that time and the risk of any laparotomy. At present, uncomplicated patency should not present any serious difficulty to the competent surgeon. The mortality in cases in which prolapse has occurred depends on the duration of obstruction, so that a uniform comment cannot be made.

CLINICAL HISTORY

Some abnormality of the umbilical cord is usually noticed at birth. It may be unusually large or slow in healing. A red, moist, granulomatous mass is generally present in the umbilicus at all times. It may bleed easily when traumatized. In the center of this mass a small aperture can usually be detected, from which some drainage may occur. The drainage may be mucoid in character, or it may have a fecal appearance, with occasional partially digested food particles. The drainage is alkaline in reaction unless there is heterotopic gastric mucosa present, in which event it is acid. The periumbilical skin will be excoriated from this drainage unless painstaking nursing care is instituted. Following feedings or during crying or straining, the drainage may be increased. However, in some instances the only drainage at any time is a clear mucous material. When some drainage occurs before the cord structures have sloughed away, one may mistake the condition for an infection of the cord. Rarely the umbilical fistula may be of such size that all the fecal material is drained out at this point.

DIAGNOSIS

The history of some abnormality of the cord at birth, more frequently in a male infant, followed by drainage, and then the appearance of a red mass in the umbilicus after the cord structures have sloughed away are suggestive of a patent omphalomesenteric duct. Fecal drainage is almost pathognomonic. The introduction of a probe into the aperture in the red mass does not establish the diagnosis and could possibly lead to serious complications, such as perforation of the bowel within the abdominal cavity. The most satisfactory diagnostic procedure is the introduction of radiopaque material through a small catheter into the aperture. Subsequent roentgenologic examination will visualize the short communication which extends down to the ileum

from the anterior abdominal wall and conclusively establish the diagnosis. Meckel's diverticulum without umbilical patency has been roentgenologically demonstrated.[49]

The patent omphalomesenteric duct must be distinguished from granulation tissue in the stump of the umbilical stalk, due to infection. A superficial remnant of omphalomesenteric duct may be present, which presents the same appearance at the umbilicus as the completely patent duct but which does not communicate with the intestine. In this type of anomaly there may be a mucous secretion which is similar to that in some of the cases of patent duct. Roentgenologic examination with radiopaque material differentiates this condition, as none of the substance enters the intestine. A patent urachus with or without urinary discharge may be confused with the patent omphalomesenteric duct. Urinary secretion at the umbilicus is diagnostic of patent urachus.[50] Another condition which may simulate the patent omphalomesenteric duct occurs as the result of ligation of a loop of bowel which may have prolapsed into the umbilical cord. After the ligature on the cord has sloughed, an intestinal fistula may develop or the infant can succumb to intestinal obstruction. Such cases have been reported by Stadtfeld, Schneider, and Thutichum.[51] A complete fecal fistula of the umbilicus was formerly more likely due to faulty ligature of the cord than to the existence of a patent omphalomesenteric duct. One instance of an appendical fistula has been reported by Jordan,[52] in which the appendix was caught during ligation of the umbilical cord.

TREATMENT

Once the diagnosis of patent omphalomesenteric duct has been definitely established, the surgical treatment should be carried out at the earliest feasible moment, because of the relatively high incidence of prolapse of the intestine which occurs through the umbilical fistula. When this complication occurs, there is obviously more risk involved. This risk is dependent on the length of time which the prolapse exists before treatment.

In cases without complications the umbilicus should be excised by an elliptic incision, the fascia separated on either side and the peritoneum

49. Buckstein, J.: Clinical Roentgenology of the Alimentary Tract, Philadelphia, W. B. Saunders Company, 1940, p. 329. Case J. T.: Jejuno-Ileal Diverticula, Acta radiol. **6**:230, 1926. Dixon, J. F.; Deuterman, J. L., and Weber, H. M.: Diverticula of the Intestine, Surg., Gynec. & Obst. **66**:314, 1938. Pfahler, G. E.: The Roentgenologic Diagnosis of Meckel's Diverticulum, ibid. **59**:929, 1934. Prevot, R.: Meckelschen Divertikel im Rontgenbild, Röntgenpraxis **8**:397, 1936.

50. Atcheson, O. W.: Patent Urachus, with a Report of Two Additional Cases, J. Urol. **33**:408, 1935. Cross, J. B.: Cyst of Urachus, J. Urol. **33**:408, 1935.

51. Schneider, von Stadtfeld and Thutichum, cited by Neff.[3]

52. Jordan, cited by Neff.[3]

opened. The short segment of duct should be resected at its junction with the ileum, a small "cuff" of duct being left which can be closed longitudinally. If there is any apparent encroachment on the lumen as a result of the closure, a side to side anastomosis should be carried out about the point of closure.

When the intestine has prolapsed out through the umbilical fistula, it is possible to reduce the prolapse manually if the infant is seen soon. After the prolapsed bowel has become edematous, it will be extremely difficult to reduce this by manual means. When it is possible to obtain reduction of the prolapse, then the operation should be performed as outlined for the condition without complications as soon as possible. When the prolapse cannot be reduced then one must proceed with a resection of the umbilicus, the duct and the involved portion of bowel with a side to side anastomosis of the afferent and efferent loops.

Ether is probably the most satisfactory anesthetic. One may minimize the amount of ether required by infiltration of a 0.5 per cent solution of procaine hydrochloride about the umbilicus and in the fascia. Infusion of isotonic solution of sodium chloride is generally necessary during the operation, and if the prolapse has been of several hours' duration it may be necessary to give a transfusion of plasma or blood. The most skilful surgical treatment cannot make up for the hazard of shock due to a deficit in volume of circulating fluid. This can occur from loss of plasma-like fluid from the prolapsed and partially strangulated loops of bowel. The use of penicillin, sulfonamide compounds or both tends to minimize the dangers of postoperative infections.

SUMMARY

1. A presentation of the history, embryology, anatomy and pathology, heterotopia, relation to other congenital anomalies and sex incidence of the diverticulum of Meckel is given. The relationship between it and the patent omphalomesenteric duct is discussed.

2. One hundred and two cases of patent omphalomesenteric duct and 26 additional cases in which prolapse of the intestine has occurred through this structure have been collected from the literature. These are briefly summarized.

3. Two new cases of uncomplicated omphalomesenteric duct and 1 case with prolapse are presented.

4. The clinical features, diagnosis and treatment of patent omphalomesenteric duct are discussed.

5. Operation for excision of the patent omphalomesenteric duct should be undertaken as soon as the condition is diagnosed, to avoid the hazard of intestinal prolapse.

GRANULOMAS CAUSED BY SURGICAL SILK AND COTTON SUTURES

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INFECTION in clean wounds following the use of surgical silk or cotton sutures is relatively uncommon when careful technic is employed. When infection does occur, it is usually minimal and produces a small stitch abscess or sinus tract. In rare cases a late infection develops several months following the primary operation, which forms a small abscess about one or more sutures surrounded by granulation tissue and a dense wall of fibrous tissue.

The following 5 cases are examples of granulomatous lesions which developed after the use of surgical silk and cotton in clean wounds (fig.).

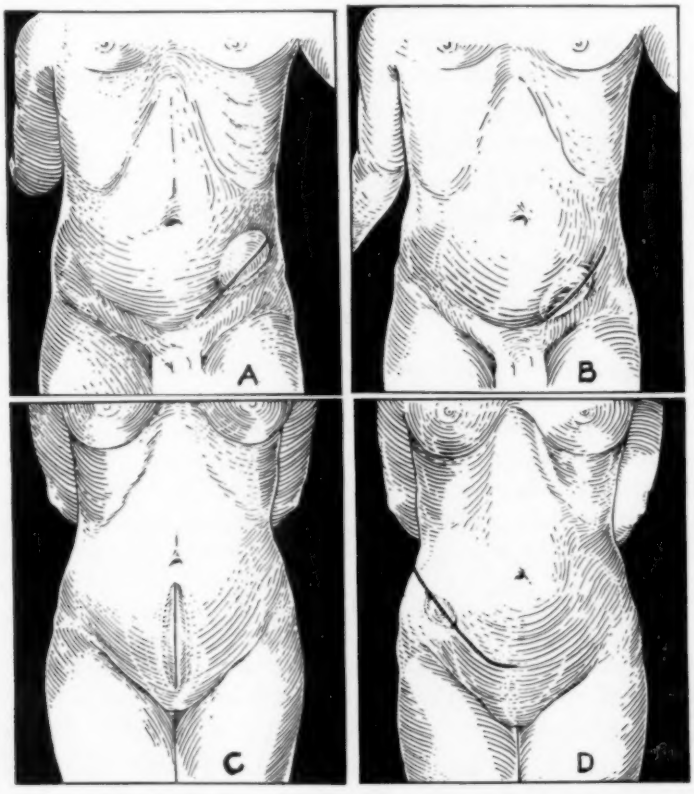
REPORT OF CASES

CASE 1.—H. V., a youth aged 19, was operated on March 6, 1941, for an indirect inguinal hernia on the left side. Size 000 interrupted surgical silk U. S. P. was used. Size 0000 surgical silk was used for ligatures. The wound healed per primam. Two months later some discomfort was noted in the region of the scar. A few days following the onset of the discomfort a swelling appeared beneath the lateral end of the scar. The swelling was tender, but pain was never severe. The patient was readmitted to the hospital July 3, 1941, approximately four months after his operation. A mass 6 by 8 cm. was visible and palpable beneath the lateral half of the scar. An incision was made through the scar, and a sinus tract was found extending from the aponeurosis of the external oblique muscle to the purse string suture used to ligate the neck of the hernial sac. An abscess 2 by 2.5 cm. surrounded the purse string suture. The purse string suture and two small sutures were removed from the abscess and sinus tract. A hemolytic staphylococcus was grown from a culture of pus..

CASE 2.—R. S., a woman aged 28, was operated on Sept. 17, 1942, for an intestinal obstruction following a pelvic operation. The obstruction was released, and a Witzel enterostomy was done above the site of obstruction. The enterostomy tube was sutured in the wound. The fasciae were closed with size 000 surgical silk. Size 0000 surgical silk was used for ligatures. The wound healed per primam. A sinus tract persisted after removal of the enterostomy tube on the eleventh postoperative day. The sinus tract did not heal until a stitch was removed six months after the operation. In December 1943, approximately fifteen months after the operation for intestinal obstruction, a slightly tender swelling was noted beneath the scar. The swelling persisted until the patient's admission to the

hospital on June 25, 1944, twenty-one months after the operation. There was a mass beneath the abdominal scar measuring approximately 5 by 10 cm. The mass was firm and slightly tender. On exploration a small abscess cavity, surrounded by a thick fibrous wall, was found extending along the suture line in the fascia, which was filled with thick pus and granulation tissue. Nine surgical silk sutures were removed. Healing was prompt after removal of the surgical silk sutures and drainage.

CASE 3.—A woman aged 60 was operated on Dec. 29, 1944. A colectomy on the right side was done through an oblique incision in the lower part of the abdomen. The wound was closed in layers with size 000 surgical silk. Size 0000 surgical silk was used for ligatures. A drain was placed in the lateral end of



Locations of granulomas which developed from two to twenty-two months following the primary operations. *A* and *B*, inguinal hernias repaired. *C*, suprapubic incision for postoperative intestinal obstruction. *D*, oblique incision for resection of right side of the colon.

the incision. In August 1945, eight months after the operation, a painless swelling was noticed by the patient beneath the lateral third of the scar. She was readmitted to the hospital, and a mass involving the scar, 4 by 5 cm., was excised on Oct. 27, 1945, ten months after the primary operation. The mass was com-

posed of dense fibrous tissue, in the center of which a small abscess was found. Surgical silk sutures were found in the wall of the granulation tissue of the abscess. A culture of the pus from the abscess grew Escherichia coli. The report on examination of the tissue was acute and chronic granulation tissue and foreign body reaction.

CASE 4.—A. F., a man aged 67, was operated on March 15, 1944, for direct, indirect and femoral hernias on the left side. The three hernias were repaired with a McVay type of operation. Size 40 interrupted cotton sutures were used. Size 60 cotton was used for ligatures. The wound healed per primam. On Jan. 13, 1946, twenty-two months after the operation for hernia, this patient was readmitted to the hospital, complaining of a tender swelling in the left inguinal region. The swelling and tenderness were first noticed two weeks before admission to the hospital. When examined, the swelling measured about 6 by 8 cm. It lay directly beneath the hernial scar. The entire area involved was indurated and tender. At operation an abscess containing thick pus and granulation tissue was drained. The abscess extended from the suture line in the aponeurosis of the external oblique muscle to the suture line along Cooper's ligament. Four cotton sutures were removed from the granulation tissue along the suture line in the external oblique aponeurosis. A hemolytic streptococcus was grown from a culture of the pus.

CASE 5.—J. T. E., a man aged 50, was operated on for appendicitis in a hospital on the West Coast four years prior to his admission to the University of Kansas Hospitals on Aug. 9, 1946. Three months before admission pain and tenderness developed beneath his abdominal scar following heavy lifting. During the three month period before his admission to the hospital, the tenderness persisted and a small mass developed at the site of his appendectomy. He was able to continue his work. On examination a slightly tender mass was found beneath the scar which measured about 3 by 6 cm. It was not attached to the skin and lay deep in the abdominal muscles. The temperature, pulse rate and leukocyte count were normal. At operation an abscess 2 by 4 cm. was found involving the transversalis and internal oblique muscles. The wall of the abscess was about 0.5 cm. thick. Several surgical silk stitches were found in the wall of the abscess. The operating surgeon reported that the surgical silk used in the muscle and fascia was size 1. Culture from the wound showed an anaerobic gram-positive bacillus of a type not identified.

NOTE.—This patient was operated on after the sketches of the first 4 cases were made.

SUMMARY

Late infection caused by nonabsorbable sutures usually develops slowly. Pain is not severe, and the patient may complain only of discomfort. A firm indurated mass forms beneath the scar of operation, which is usually slightly tender. There may be a slight rise in temperature and leukocyte count. When incised, a relatively small abscess is found surrounded by a firm wall of fibrous tissue 1 cm. or more in thickness.

Granulomas caused by surgical silk and cotton must be uncommon, since they occurred but four times in several hundred clean operations in the University of Kansas Hospitals. Shambaugh [1] recorded 1 case in which a secondary abscess developed after the use of surgical silk five months after complete healing. Leithauser [2] stated that he has abandoned the use of nylon as suture material, since granulomas may form as late as ten months after its use.

1. Shambaugh, P.: Postoperative Wound Complications, Surg., Gynec. & Obst. **64**:765 (April) 1937.

2. Leithauser, D. J.: Confinement to Bed for Only Twenty-Four Hours After Operation, Arch. Surg. **47**:203 (Aug.) 1943.

STRANGULATED INGUINAL HERNIA

Observations in Fifty Cases

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IN THIS article, based on the observations that I have been able to make on the 50 patients with strangulated hernia that came under my care during the past few years, an attempt is made to describe the morbid anatomic evolution of a strangulated hernia. Of these 50 cases, 12 cases are illustrated, as each one of these depicts a phenomenon of certain pathologic significance. An analysis of the important findings, not involving biochemical or bacteriologic investigations, is also included in the article.

The anatomic changes that are brought about as a result of the constriction which gradually leads on to strangulation affect (1) the mesentery, (2) the blood vessels in the mesentery and in the bowel wall and (3) the wall of the intestine itself.

From the observations based on the changes that take place in these three fundamental regions, an attempt is made to illustrate the morbid anatomic processes of the disease and to try and correlate these changes which occur in different parts and also to arrange these in their chronologic sequence.

A study of the illustrations clearly shows that the morbid anatomic changes are not confined to the bowel wall alone but are usually present in the attached mesentery also of the affected loop. In fact, I believe that the changes in the mesentery precede those in the intestinal wall. The illustrations depict (1) the seven stages in the evolution of a strangulated hernia, in their chronologic order, from the stage of mere congestion to one in which the intestinal loop becomes nonviable; (2) the pathologic appearances in the formation of a hematoma, from the stage of dilatation of the veins in the mesentery to the stage when a hematoma may occupy a large portion of the mesentery, and (3) the relation between the changes that take place in the mesentery and the bowel wall.

Thus the following changes are noted: 1. The earliest stage is that in which a normally creamy pink color of the bowel wall, usually the small intestine, changes to one in which it appears flushed (fig. 1 *A*).

A part of the paper was read before the staff meeting of the Grant Medical College and Sir Jamsetjee Jejeebhoy Group of hospitals.

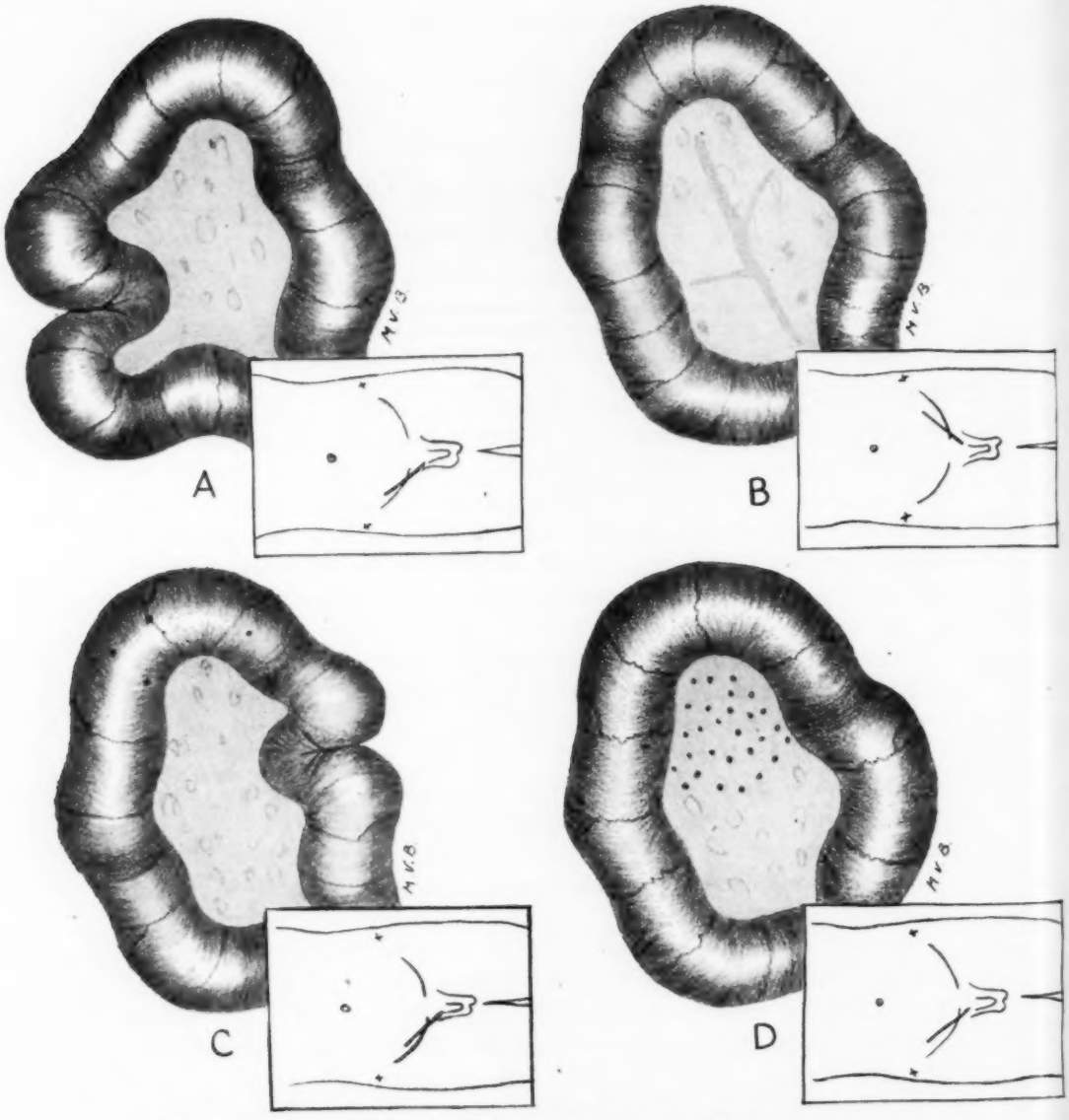


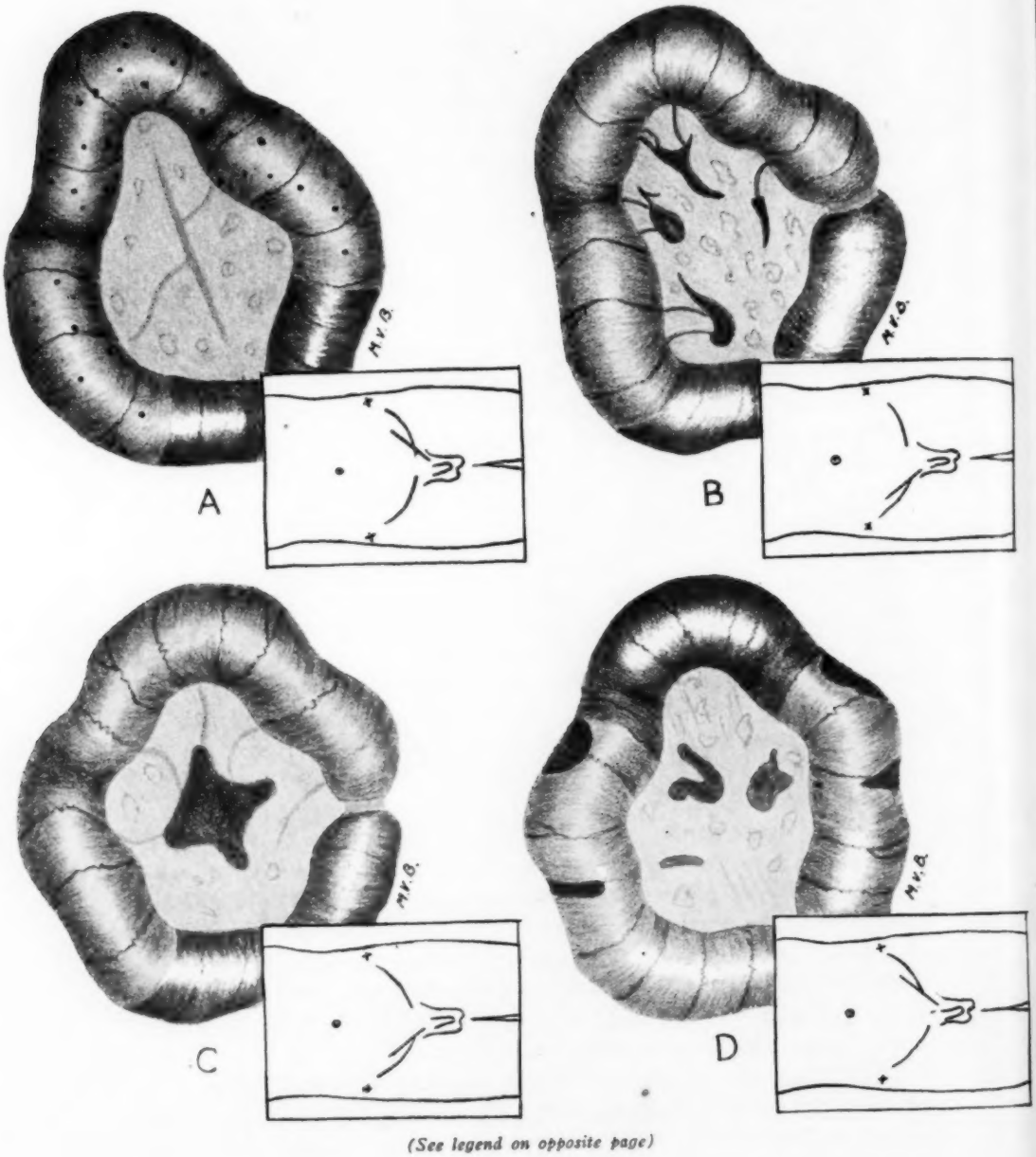
Fig. 1.—Evolution of strangulated hernia. *A* (case 5633 of 1938), stage 1. A loop of small intestine flushed in appearance is seen. It has lost its usual creamy pink color. The blood vessels coursing on the bowel wall are clearly visible. There is no mark of constriction, the site of strangulation. *B* (case 104 of 1940), stage 2. The illustration shows a loop of small intestine which is cyanosed in appearance. The blood vessels on the bowel wall are clearly seen. The mesentery is thickened, and the veins in the mesentery have become visible owing to dilatation. This is the first stage in the formation of a hematoma. *C* (case 5527 of 1938), stage 3. A loop of intestine flushed in appearance. The blood vessels on the bowel wall are clearly visible. A few hemorrhagic spots are seen on the bowel wall. *D* (case 408 of 1939), stage 3. A loop of small intestine. The loop has lost its normal color and appears flushed. The blood vessels on the bowel wall are prominently seen. The mesentery shows a peculiar appearance; it shows numerous dotlike hemorrhagic spots. These dots in the course of time would have gone to form a hematoma. This shows the second stage in the formation of a hematoma, the first stage being dilatation of veins as seen in *B*.

2. When the obstruction to the circulation has persisted a little longer, the veins in the mesentery appear dilated and there is thickening of the mesentery, with cyanotic appearance of the bowel wall (fig. 1 *B*). 3. When the obstruction has continued a little longer, the edema of the mesentery appears to persist, and, in addition, hemorrhagic spots appear either in the bowel wall or in the mesentery, but cyanosis may be absent (fig. 1 *C* and *D*). 4. At a later stage the hemorrhagic spots appear in the cyanosed part of the loop (fig. 2 *A*). 5. At a still later period, there may be present a central hematoma in the mesentery or small hematomas may appear along the line of the blood vessels in the mesentery. The bowel wall corresponding to this portion of the mesentery always shows pathologic changes (fig. 2 *B, C* and *D*). 6. Still later the cyanosis in the bowel wall becomes more striking, with appearance of blue-black bands along the circumference of the bowel wall in the line of the blood vessels, or the bowel wall may appear brown-mauve-purple or chocolate colored or like wet blotting paper. The hematoma in these cases is either large or peripheral (fig. 3 *A, B* and *C*). 7. When the strangulation has reached its final stage, one finds the typical classical picture, as usually described in the textbooks on surgery, of a nonviable loop of intestine. In the particular case illustrated, the mesentery is seen to be almost completely occupied by a large flame-colored hematoma (fig. 3 *D*).

It is interesting to observe that in every case the bowel wall that appears affected always corresponds to the portion of the mesentery which is also affected. It is noteworthy that the margin of the hematoma and the line of demarcation of the affected and normal parts of the loop always stop short sharply at the line of constriction. It is also worth observing that whenever the gray band of constriction was seen in this series of cases it was present on only one limb of the loop.

One may therefore conclude that the seven stages in the evolution of a strangulated hernia are (1) the stage of congestion; (2) the stage of cyanosis with edema of the mesentery and visible dilated veins; (3) the stage of appearance of hemorrhagic spots; (4) the stage of cyanosis with hemorrhagic spots; (5) the stage of cyanosis of the bowel wall with a hematoma in the mesentery; (6) the stage of deeper cyanosis, with blue-black bands in the wall of the intestine or rough, wet, blotting paper appearance with a large or peripheral hematoma in the mesentery, and (7) the stage of nonviable loop.

The study of several illustrations suggests, as already stated, the working of another interesting pathologic process. In all the illustrated cases it will be observed that whatever may be the changes in the bowel wall they are invariably confined only to the part of the intestinal loop whose draining veins, while coursing through the mesentery, have to pass through the area of the mesentery which is occupied by the hema-



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toma. The sequence of events is therefore likely to occur in the following order:* 1. Constriction at the external or internal ring causes obstruction to the venous channels, and dilated veins appear in the mesentery (figs. 1 *B* and 2 *A*). 2. Due to overfilling of the veins, there is an exudation of serum into the mesentery, giving rise to edema, which on the operating table is recognizable as thickened mesentery. 3. Continuous overfilling and blood stagnation ultimately cause rupture of the wall of the veins, giving rise to hematomas, which in the early period are limited to the line of the blood vessels—the venous channels—and in their extremely early period probably appear like small hemorrhagic spots (figs. 1 *D*, and 2 *B*, *C* and *D*). 4. At a later stage the hematomas increase in size and tend to coalesce and form a big hematoma, which then occupies an area varying in size. The hematoma causes further pressure on the easily compressible veins and gives rise to thrombosis (fig. 3 *A*, *B* and *C*). 5. Once thrombosis has occurred, further deterioration in the condition of the bowel wall is likely to be rapid.

Clinically, as a guide to the surgeon while operating on patients with strangulated inguinal hernia, I believe that all cases can be divided into three groups.

1. One group consists of cases in which, on opening the sac, only the color of the bowel wall is found to be slightly altered, the bowel being normal in every other way and peristalsis present as in the normal

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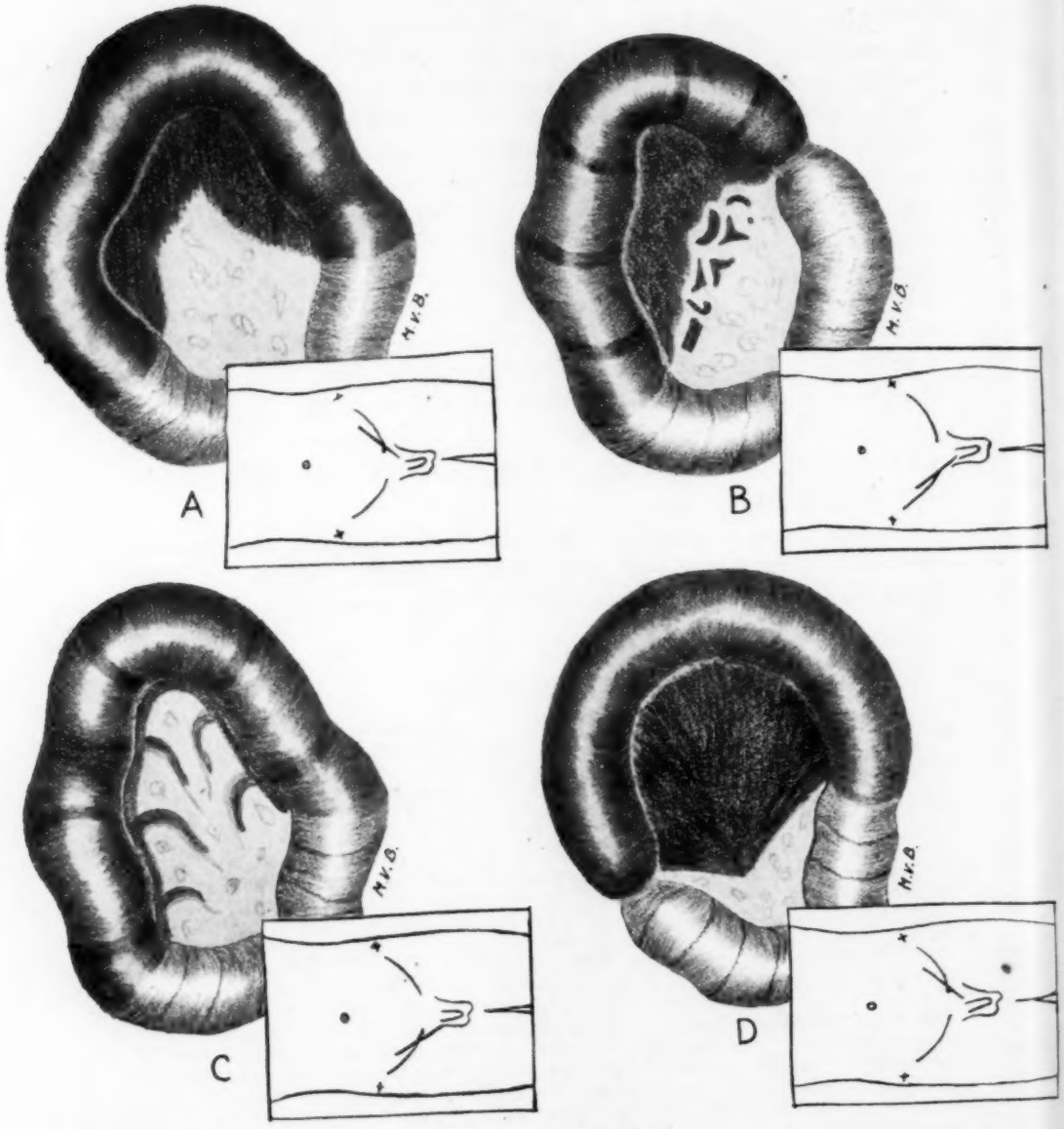
EXPLANATION OF FIGURE 2

Evolution of strangulated hernia depicted. *A* (case 5617 of 1938), stage 4. A cyanosed loop of small intestine. The blood vessels are clearly visible on the bowel wall. Numerous dotlike hemorrhagic spots are seen on the bowel wall but only on the portion which has a cyanosed color. There is no mark of constriction, the site of strangulation. *B* (case 95 of 1941), stage 5. A cyanosed loop of intestine. The blood vessels are clearly visible on the whole loop. On one limb of the loop is a grayish colored band, the site of strangulation, showing a constriction and demarcating the normal from the affected part of the loop. There is no mark of constriction on the other limb of the loop. In the mesentery attached to the cyanosed part of the loop there are several prominent blood vessels—both arteries and veins. Toward the visible proximal end of these blood vessels are seen formed four various-shaped small hematomas. The rest of the mesentery is normal in appearance. This is the third stage in the formation of a hematoma. *C* (case 1352 of 1942), stage 5. A loop of small intestine cyanosed in appearance. The blood vessels are clearly visible on the whole loop. On one limb only of the loop is a grayish colored band, the site of constriction and strangulation. In the mesentery is seen a central hematoma. The veins draining the cyanosed part of the intestine, which have to pass through this region, have become dilated because of obstruction and are therefore visible. This is the third stage in the formation of a hematoma. *D* (case 3130 of 1940), stage 5. A loop of small intestine, parts of which appear cyanosed. On the parts which are cyanosed are seen patches of ecchymosis. The blood vessels are clearly visible on the whole loop. In the mesentery are scattered hematomas, and the affected part of the intestine corresponds to the site of the hematomas, showing that the cyanosis of the parts of the intestine is likely due to the obstruction of the veins which have to pass through the region of the hematomas. This is again the third stage in the formation of a hematoma.



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intestine. In such cases the intestinal loops may be immediately and safely returned into the abdominal cavity.

2. Another group consists of cases in which there are changes in the bowel wall and/or in the mesentery—changes affecting the color and condition of the bowel wall in varying degree and changes in the mesentery varying from its mere thickening to a large hematoma. When the changes are slight, the normal color returns within a short time of relieving the constriction and application of packs of warm isotonic solution of sodium chloride. In more advanced cases it may take several minutes before any appreciable change in the color may be detected. Also it may take some time for the peristalsis to reappear; but once the peristalsis has definitely begun, no matter what the color, feel or odor of the bowel wall and/or of the mesentery may be, so long as reinduced persistalsis persists and there is no perforation, the loop may confidently be returned to the abdominal cavity. In fact, I feel that the presence or absence of peristalsis is the only sure token of the viability or non-

EXPLANATION OF FIGURE 3.

Evolution of strangulated hernia depicted. *A* (case 6339 of 1938), stage 6. A loop of small intestine. The loop is reddish brown, chocolate brown or like wet blotting paper. It has lost its gloss and sheen and appears mat and sticky. The surface is rough. The blood vessels are not visible on this affected part of the loop but can be seen on the end of the limbs of the loop. The rough appearance is due to wide ecchymosis. The mesentery toward its attachment to the affected part of the loop is occupied by a large brownish hematoma, which has acquired the bend of the loop. This again shows the effect of the hematoma in producing changes in the bowel wall. The rest of the mesentery is normal in color. There is no gray band which is suggestive of the site of strangulation. *B* (case 811 of 1940), stage 6. A loop of small intestine, part of which is deeply cyanosed with blue-black bands at intervals along its circumference in the line of the blood vessels. The blood vessels on the affected loop are not visible. There is a mark of constriction on one limb only of the loop. The mesentery is occupied by a large hematoma, with a few scattered hematomas proximal to the large hematoma. In the course of time, if the strangulation had continued, these scattered small hematomas would have coalesced with the big hematoma. This is the fourth stage in the formation of the hematoma. *C* (case 6578 of 1941), stage 6. A loop of small intestine, part of which is deeply cyanosed with blue-black bands along its circumference in the line of the blood vessels. The blood vessels are not visible on this part of the loop. There is no mark of constriction, which is suggested by the appearance of a gray band. In the mesentery there is a peripheral hematoma—a hematoma of a linear type along the intestinal attachment of the mesentery. There are conical prolongations proximalward into the mesentery from the linear hematoma. Dilated veins are visible along these projections from the hematoma. This is a hematoma which from its size and shape belongs to the third stage of hematoma formation and fifth stage in the evolution of strangulated hernia, but its peripheral disposition has produced changes of a later stage. *D* (case 13 of 1940), stage 7. A loop of small intestine, part of which is nonviable. This part is rough, sticky and blue-black in color. It is uniform in outline and shows complete absence of peristalsis. The blood vessels are not at all seen on this part of the loop. A mark of constriction, as suggested by the presence of a gray band, is seen on one limb only of the loop. The mesentery attached to the nonviable part of the loop is occupied by a large flame-colored hematoma. This is the final, or seventh, stage in the evolution of a strangulated hernia and the final, or fifth, stage in the formation of a hematoma.

viability of the bowel wall and that it is the only sure criterion as to whether the intestines should be returned into the peritoneal cavity.

3. In the third group are included the cases of strangulation in which the loop does not regain its peristaltic power and must therefore be considered as nonviable, even if the color and other changes are not those typical of a nonviable loop. In such cases the appropriate surgical procedure must be carried out.

All the 50 patients were operated on under spinal anesthesia, it being induced in some with 1.2 to 1.5 cc. of 5 per cent stovaine and in others with 10 cg. of procaine hydrochloride dissolved in the patient's cerebrospinal fluid. After the operation, all patients complained of difficulty in passing urine for a period varying from twenty-four to seventy-two hours after the operation, and all had to be catheterized.

All patients were men.

The ages of the patients varied from 20 to 82 years. The largest number of cases (26) occurred between the ages of 31 and 50 years.

TABLE 1.—*Age Groups of Patients with Strangulated Inguinal Hernia*

Age, Yr.	Number	Per Cent
Below 21	1	2
21 - 30	11	22
31 - 40	14	28
41 - 50	12	24
51 - 60	8	16
Above 60	4	8

There was only 1 patient below 21 years. Table 1 gives the different age groups.

All the hernias were of the indirect inguinal type, and the majority of these (29, or 58 per cent) were of the right side. There were no cases of bilateral strangulation.

Of the 50 cases, in 44 cases the hernial sac contained fluid, in 4 cases there was no fluid and in 2 cases it was not recorded. Of the 44 cases, in 20 cases the fluid was of the serous type while in 24 cases the fluid was definitely sanguinous in nature.

In the majority of the cases (36, or 72 per cent) the sac contained loops of small intestine; it contained omentum alone in 3 cases, omentum and small intestine in 7 and large intestine in 2. In 2 of the cases the contents could not be identified with certainty, as the hernia was spontaneously reduced before the operation but after the spinal anesthesia was given. Table 2 gives the comparative figures.

Of the 50 patients, 5, i. e., 10 per cent, died before their discharge from the hospital. Postmortem examination was not available in any of the 5 cases. One patient, the patient who was 82 years old, died

suddenly on the eighth day after the operation, the clinical diagnosis given being coronary thrombosis. One pateint, in whom the resection of the nonviable loop with drainage through Paul's tubes was done, died within twenty-four hours after the second operation of end to end anastomosis, from continuously increasing abdominal distention. The second operation was done eleven days after the first operation. One patient died within a few hours of the operation. Two patients died two and four days after the operation. Both had nonviable loops of small intestine, and in both patients excision of the loops with drainage through Paul's tubes was done. It was observed during their postoperative life that there was an enormous quantity of greenish fluid which drained from Paul's tubes.

When the patient who died of coronary thrombosis is excluded, one can therefore say that only 4 patients died of strangulated inguinal hernia, a mortality rate of 8 per cent.

TABLE 2.—*Comparison of Contents in Hernial Sac*

	Present Series, Approximate per Cent	Maingot, R.: Post-Graduate Surgery, London, Butterworth & Company, 1936, vol. 1, p. 1037 Approximate per Cent	Frankau, C.: Brit. J. Surg. 19 : 176, 1931, Approximate per Cent
Small intestine alone or with omentum.	86	85	75
Large intestine.....	4	6	5
Omentum alone.....	7	8	6.2

Of the 50 cases, in only 4 cases there was not a history of hernia. In these 4 cases, i. e., 8 per cent, the first appearance of the hernia was manifested by the advent of strangulation. In 3 other cases a complete history is not recorded; 8 patients had hernia of less than one year's duration, 11 patients had hernia for a period varying from one to three years and 7 patients from three to five years, while 17 patients had hernia of more than five years' duration. Of the 17 cases, the hernias in 4 cases were of a recurrent type.

One patient had an unusual postoperative complication. During the operation, after the strangulation was relieved and the intestines were being returned into the abdominal cavity, the patient passed a large hemorrhagic stool and suddenly collapsed, with stoppage of respiration and loss of consciousness. Artificial respiration was immediately started and nikethamide given intravenously. While artificial respiration was being given, one of the lower ribs on the left side was accidentally fractured. Another injection of nikethamide was given, by the intracardiac route. Artificial respiration had to be continued for three quarters of an hour, the patient remaining unconscious all that time. He then

TABLE 3.—*Review of Cases of Strangulated Inguinal Hernia from 1938 to 1942*

Reg. No.	Name	Age, Yr.	Duration	History of Hernia	Signs and Symptoms	Side	Nature of Fluid	Contents of the Sac	Comment
					1938				
5527 (fig. 1 *C*)	B. A.	45	6 days	A few months	1. Vomiting for the past few days	Right	Large amount of serous fluid	The loop of bowel showed a few subserous hemorrhagic spots	Apex of the sac was adherent to the top of the testicle; numerous thick adhesions; obstruction on the same side some months previously
5593 (death)	M. D. S.	82	2 hr.	6 mo.	1. Patient looked extremely ill	Right	No fluid	Not noted	Three attacks of irreducibility in the last 6 mo.; patient suddenly collapsed and died on the 8th day of operation; hernia operation done on the left side in 1919
5617 (fig. 2 *A*)	G. V.	38	7 hr.	3 yr.	1. General condition good 2. Pulse slow but feeble 3. Painful inguinal swelling	Left	Fair amount of brownish fluid	Part of the loop was cyanosed in color, and this showed numerous points of subserous hemorrhages; dilated veins in the mesentery	Three attacks of irreducibility
5633 (fig. 1 *A*)	S. J.	55	6 hr.	20 yr.	1. General condition good	Right	Fair amount of brownish fluid	Two short congested loops of small intestine	1. Immediately after reducing the bowel the patient passed a large foul-smelling stool, collapsed and became unconscious; artificial respiration was done, during which 1 lower left rib was fractured 2. Unconscious for ¾ hr. 3. For 10 days after operation he was mentally abnormal then gradually recovered 4. Attacks of irreducibility twice during the last 6 mo.
5718	D. S.	50	1 day	5 mo.	1. Vomited 3 times 2. Painful swelling	Right	Fair amount of yellowish fluid	Loop of small intestine	
6339 (fig. 3 *A*)	G. S. P.	30	8 hr.	1 yr.	1. General condition good	Left	Small quantity of sanguinous fluid	1. Color of the obstructed loop cyanosed mauve-purple like wet blotting paper 2. Ecchymosis in the mesentery, near its attachment to the bowel	
6366	F. L.	60	3 hr.	About 1 yr.	1. General condition good	Right	No fluid in sac	1. Small mass of omentum 2. No bowel	Sac was particularly thin and friable
49 (Bhajekar Memorial Hospital)	D. P. B.	73	6 hr.	10 yr.; recurrence	1. General condition good 2. No vomiting 3. Painful inguinal swelling	Right	Fair amount of serous fluid	1. One loop of small intestine 2. One loop of large intestinal cecum	Sliding hernia; patient was operated on for strangulated right inguinal hernia 10 yr. ago; during the last 10 yr. he has had several attacks of irreducibility lasting for 1 hr. or so
					1939				
405 (fig. 1 *D*)	M. K.	30	3 hr.	7 yr.	1. General condition good 2. Considerable local pain 3. No vomiting	Right	Small amount of serous fluid	Two loops, each about 1 ft. in length, with a large area of mesen-	An attack of irreducibility 2 yr. ago but reduced

Memorial Hospital)					3. Painful inguinal swelling				had several attacks of irreducibility lasting for 1 hr. or so

1939

405 (fig. 1 *D*)	M. K.	30	3 hr.	7 yr.	1. General condition good 2. Considerable local pain 3. No vomiting	Right	Small amount of serous fluid	Two loops, each about 1 ft. in length, with a large area of mesentery drawn in; part of the mesentery was edematous, and on both sides there were hemorrhagic spots	An attack of irreducibility 2 yr. ago but reduced
438	M. K.	40	8 hr.	6 mo.	1. General condition good 2. No vomiting 3. Pain in the abdomen	Left	Small amount of sanguinous fluid	One small loop of bowel cyanosed in color; 1 linear mark on 1 limb at the site of construction	
1711 (death)	M. W.	54	4 days	6 yr.	1. General condition bad 2. Vomiting almost every hour for 2 days 3. Absolute constipation 4. Tongue dry 5. Hiccup 6. Painful inguinal swelling	Left	Small amount of sanguinous fluid	1. About 1 foot length of small intestine cyanosed in color and no peristalsis even after bath in hot isotonic solution of sodium chloride 2. A mass of gangrenous omentum	1. After ligation of mesenteric vessels, gangrenous loop of intestine was excised; 2 Paul's tubes were tied into the 2 limbs; 9 days later, another surgeon did end to end anastomosis; within 24 hr. the patient had distention and died 2. Gangrenous mass of omentum was also excised
2021	M. R.	32	1 day	.....	General condition good	Left	Small amount of sanguinous fluid	Two loops of congested and cyanosed intestine, each about 9 in. long; mesentery of the two loops was thick and discolored	
3805	A. W. S.	50	21 days	3 mo.	1. General condition good 2. No vomiting 3. Tense painful swelling	Right	Small amount of sanguinous fluid	One loop of congested bowel	Hernia developed while patient had a severe cough
4131	J. M.	40	4 days	10 yr.	1. General condition good 2. Constipation 4 days 3. No vomiting	Right	Fair amount of sanguinous fluid	Sac contained cecum and appendix	Appendix was removed
5647 (death)	D. G.	38	6 hr.	1 yr.	1. General condition bad 2. Vomited 2 times 3. Tense painful swelling 4. Tongue dry 5. Patient perspiring profusely	Left	Fair amount of sanguinous fluid	Two ft. of small intestine 18 in., nonviable 1. Color was dark blue-violet 2. Bowel had lost its sheen 3. Loop felt sticky and heavy 4. Foul smell 5. Complete absence of peristalsis	After ligation of the mesenteric vessels, the nonviable loop was excised; 2 Paul's tubes were tied in the 2 limbs; patient died on the 4th day
7294	H. Y.	60	2 hr.	20 yr.	1. Painful inguinal swelling 2. No vomiting but tongue dry	Right	No fluid in the sac	Loop of small intestine	Irreducible since 9 p. m. on 12/18/39; it was reduced by taxis; became irreducible again on 12/20/39 and was operated on immediately; the hernia had 3 sacs—the outer 2 of fibrous tissue and the innermost of peritoneum; the middle sac had developed yellowish mucoid cysts in its wall
7295	I. N.	40	5 hr.	2 yr.	1. General condition good 2. Vomited once; tongue dry and coated 3. Complete constipation	Right	No fluid in the sac	One loop of normal small intestine	"nominal."
7382 (death)	R. M.	45	1 day	Several years	1. General condition bad 2. Vomited once 3. Complete constipation 4. Semiconscious	Left	Sac contained several ounces of turbid dirty yellowish fluid	Sac contained a short loop of small intestine; mesentery along with its intestinal attachment was edematous; grayish green translucent type of edema	Patient died soon after operation

TABLE 3.—*Review of Cases of Strangulated Inguinal Hernia from 1938 to 1942—Continued*

Reg. No.	Name	Age, Yr.	Duration	History of Hernia	Signs and Symptoms	Side	Nature of Fluid	Contents of the Sac	Comment
					1940				
13 (death; fig. 3*D*)	A. R. R.	45	2 days	5 yr.	1. Frequent vomiting 2. Absolute constipation for 2 days 3. Pain in umbilical region	Left	Small amount of sanguinous fluid	1. A large mass of brownish black omentum 2. About 9 in. of nonviable loop of small intestine	All tissues at site of obstruction were flabby and friable; a lateral anastomosis was done and nonviable portion excised; wound was drained; patient died 48 hr. later
103	H. H. J.	30	9 hr.	2½ mo.	1. No vomiting 2. Painful tense swelling	Left	Fair amount of serosanguinous fluid	1. A large mass of almost black omentum 2. A cyanosed loop of small intestine about 6 in. long	The omental mass was excised
104 (fig. 1*B*)	K. L.	30	27 hr.	Since childhood	1. Vomited 10 to 11 times 2. Constipation 3. Tense tender swelling 4. Good general condition	Left	Small amount of sanguinous fluid	A short cyanosed loop of small intestine	
111	J. A.	40	8 hr.	6 yr.	1. Pain in the epigastrium 2. No vomiting	Right	Fair amount of sanguinous fluid	About 1 foot of small intestine; mucoid type of edema in the mesentery along its line of attachment to the intestine	1. Patient passed a large hemorrhagic stool immediately after operation 2. The right testicle was big, 2½ in. in diameter
112	S. P.	55	Not noted	20 yr.	1. Painful swelling 2. Vomiting 3. Constipation 4. Good general condition	Left	Fair amount of sanguinous fluid	Sac contained 2 loops of small intestine 1. One loop, cyanosed, had a conical diverticulum and patches of hemorrhage in the mesentery 2. One loop pale yellow-gray; the 2 limbs almost adherent to each other and anchored to the sac by means of a band	Both loops recovered color with bath of hot isotonic solution of sodium chloride; 4 days later pneumonia developed but patient gradually recovered; hernia gradually increasing in size for past 20 yr.
811 (fig. 3*B*)	J. K.	60	8 hr.	5 yr.	1. No vomiting 2. Good general condition	Right	Small amount of light brown fluid	1. A loop of small intestine of about 30 in., of which 9 in. was dark violet with blue-black bands; a large hematoma in the mesentery	
862	M. J.	60	16 hr.	Not noted	1. Tense tender swelling 2. No vomiting	Right	No fluid	2. Contents of sac could not be identified	1. After spinal injection the hernia reduced spontaneously
1585	D. D. K.	45	3 hr.	6 yr.	1. Vomited once	Right	Fair amount of dirty straw-colored fluid	Sac contained 2 loops 1. One loop of intestine had mucoid edema in the mesentery along its intestinal attachment 2. One loop had a patch of discoloration at its antimesenteric border	Hemorrhagic stool 3 hr. after operation
1305	P. P.	50	Not noted	Not noted	1. Patient was admitted in a semiconscious condition; he is deaf 2. Simple comminuted	Right	Small amount of serous fluid	1. Normal loop of bowel 2. Mesentery showed pronounced thickening and extremely dilated veins	

1305	P. P.	50	Not noted	Not noted	1. Patient was admitted in a semiconscious condition; he is deaf 2. Simple comminuted fracture of both calcanei 3. Simple fracture of first and second lumbar vertebrae	Right	Small amount of serous fluid	1. Normal loop of bowel 2. Mesentery showed pronounced thickening and extremely dilated veins	
2295	S. B.	35	1 day	2 yr.	1. Tense, tender, painful swelling 2. Constipation but vomited twice	Right	Small amount of sanguinous fluid	1. One ft. of congested small intestine rough on the surface like wet blotting paper	Two days after operation patient showed signs of pneumonia but recovered
2390	S. J.	24	Not noted	1 yr.	1. Painful tense swelling 2. No constipation 3. No vomiting	Left	Little serous fluid	Loops of small intestine	
3130 (fig. 2 *D*)	B. M.	20	8 hr.	No hernia previously	1. Vomited several times and constipated	Left	No fluid	Loop of bowel showed patches of cyanosis and subserous hemorrhages; mesentery was edematous and contained patches of hemorrhages	At administration of spinal anesthesia the hernia reduced; operation was, however, done and the loop brought out for inspection
3183	R. D.	70	1½ hr.	Not noted	1. Vomiting 2. Constipation	Right	Fair amount of sanguinous fluid	About 3 ft. of small intestine	
3184	H. S. R.	43	10 hr.	Several years recurrence	1. Painful swelling in the left inguinal region 2. No vomiting	Left	Fair amount of serosanguinous fluid	Small intestinal loops	Operated on 6 to 10 yr. previously for bilateral hernia
3673	M. A.	50	6 hr.	1 yr.	1. Painful swelling 2. No vomiting	Right	Fair amount of sanguinous fluid	The mesentery along the intestinal attachment at 1 place was reddish and cyanosed, and at corresponding 1 or 2 places at antimesenteric border the bowel had striations of cyanosis and of ecchymoses	
3755	S. A. H.	65	4 hr.	4 yr.	Painful inguinal swelling	Left	Small amount of serous fluid	The sac contained a large mass of omentum; in 1 place the vessels in the omentum were thrombosed and there was a small hematoma	
3766	R. K.	45	2 days	4 mo.; recurrence	1. Vomited 3 times 2. Constipated for 2 days 3. Painful tense swelling	Right	Small amount of serosanguinous fluid	Two ft. of small intestine, part of it cyanosed; mesentery of this loop was thickened and had hemorrhages in parts; the loop showed a line of previous anastomosis, the suture line being continued into the mesentery; this line was more congested than the rest of the bowel	No visible abdominal scar or scar of a previous hernia operation, but an inguinal scar about 1 in. long; history of operation 12 yr. ago for hernia

TABLE 3.—*Review of Cases of Strangulated Inguinal Hernia from 1938 to 1942—Continued*

Reg. No.	Name	Age, Yr.	Duration	History of Hernia	Signs and Symptoms	Side	Nature of Fluid	Contents of the Sac	Comment
					1940—Continued				
4615	B. B.	38	12 hr.	2 yr.	1. No vomiting 2. Tongue coated 3. Constipated 4. Painful inguinal swelling	Left	Sanguinous fluid	Contents of the sac could not be identified	Hernia reduced of its own accord just before operation, particular loop could not be identified as the one which was strangulated.
6016	S. N.	25	4 hr.	6 yr.	1. Vomited twice 2. Painful inguinal swelling	Right	Large quantity of serous fluid	Sac contained 2 ft. of small intestine; mesentery was thickened and edematous, and 2 enlarged lymphatic glands were palpable	
					1941				
242	A. B.	40	12 hr.	2 yr.	General condition good; no vomiting	Left	Large amount of dark-colored fluid	1. One small loop was dilated, had lost sheen and was grayish black 2. One loop normal in color	Five previous attacks of irreducibility; a primary side to side isoperistaltic anastomoses was done and the nonviable loop excised
814	P. A. G.	25	18 hr.	None	1. Dragging sensation around the umbilicus 2. Vomited several times during the night 3. Passed one stool	Right	Small amount of serous fluid	Distended short loop of small intestine	While sac was being opened, the coat of the loop was slightly nicked; injury buried with a purse string suture; while patient was trying to lift a heavy weight, his foot slipped and for the first time a swelling appeared in the right inguinal region and became irreducible
1681	S. G.	50	4 hr.	15 yr.	1. No vomiting 2. Painful inguinal swelling	Right	Fair amount of fluid	Sac contained 2 loops 1. 1½ ft. long; a small area in this loop bluish in color and no peristalsis 2. 2 ft. long; this loop showed dilated vessels and thickened and edematous mesentery	
4436	M. Y.	25	3 hr.	5 yr.	1. Vomited 4 times in 3 hr.	Left	Small amount of serous fluid	1. Large mass of blue omentum 2. One loop of small intestine 9 in. long, with a thickened mesentery with 2 small spots of hemorrhage 3. One congested small loop 9 in. long	Bladder was adherent to the outer surface of the medial side of the sac
6113	K. K.	40	1 day	6 yr.	Nothing particular	Left	Small amount of serous fluid	Three loops of small intestine; total length about 3 ft.	
6281	B. L.	29	6 hr.	3 yr.	1. Vomited 3 times	Right	Small amount	One short loop of small	

6113	K. K.	40	1 day	6 yr.	Nothing particular	Left	Small amount of serous fluid	Three loops of small intestine; total length about 3 ft.	
6281	B. L.	29	6 hr.	3 yr.	1. Vomited 3 times 2. Tender inguinal swelling	Right	Small amount of straw-colored fluid	One short loop of small intestine	
6277	P. J.	45	3 hr.	None	1. Pain in the abdomen 2. Painful swelling in inguinal region	Left	Small amount of straw-colored fluid	1. Large mass of omentum and 2 short loops of small intestine, each about 9 in. long	While patient was lifting weight, a swelling suddenly appeared in the inguinal region, which could not be reduced; no history of hernia
6578 (fig. 3 *C*)	B. A. M.	28	1 day	Since childhood	1. Patient looked ill 2. Vomited twice 3. Painful inguinal swelling	Right	Little fluid; straw-colored	Small loop of bowel; part of it had bluish black stripes; loop was enveloped in a mass of bluish black omentum; this mass of omentum was excised	
95 (Bhajkar Memorial Hospital) (fig. 2 *B*)	K. S. P.	55	10 hr.	23 yr.; recurrence	1. Painful swelling in the right inguinal region 2. No vomiting 3. Patient looked ill	Right	Large amount of serous fluid	1. A small tag of omentum 2. Two long loops of small intestine; 1 of the loops was purplish and the mesentery attached to this part had a few areas of hematomas of various shapes and sizes near its attachment to the intestine	During the latter part of the operation the patient collapsed, but he recovered
					1942				
1352 (fig. 2 *C*)	M. H.	40	1 day	2 yr.	1. Thirst, vomiting, constipation and hiccup 2. Swelling appeared suddenly, on straining, and became irreducible	Right	Fair amount of sanguinous fluid	One short loop, about 9 in., of small intestine with ecchymosis; 1 small hematoma on 1 side of the mesentery away from its side of attachment to the bowel	
4803	M. S.	40	4 days	2 yr.	1. General condition good 2. Tense but not tender inguinal swelling	Right	Large amount of serosanguinous fluid	Large mass of omentum and 18 in. of congested small intestine	
4942	F. T.	40	3 hr.	1 yr.	1. Vomited 3 times and had 6 watery stools 2. Tense painful inguinal swelling 3. Patient looked ill and so not inclined to talk	Right	Fair amount of serous fluid	Large length of cyanosed loop of small intestine and a large hematoma adherent to the mesentery, which could be detached	
5065	B. A.	26	3 days	3 yr.	1. No vomiting or pain 2. Tense painful inguinal swelling 3. General condition good	Left	Small amount of serous fluid	Large mass of edematous omentum	

gradually regained consciousness and the power of respiration. For ten days after the operation the patient was restless, irrelevant in speech and behavior and mentally generally deranged. From the eleventh day he gradually recovered, and he was discharged in a normal mental condition after a few days. When he was mentally normal, careful inquiry revealed that the patient remembered nothing that occurred during the abnormal period.

In 1 case the contents of the sac were of an unusual type—one loop had a short conical diverticulum. This loop was congested and cyanosed in color, with a hematoma in the mesentery. The second loop was cream gray in color, with a rough sticky surface. The bowel wall felt thickened and almost solid and was rough and cold to touch. It had lost all its gloss and sheen.

A brief description of the operation which I have uniformly adopted in this series of cases is as follows:

An incision is made beginning at a point above the level of the surface marking of the internal abdominal ring. It is extended obliquely downward and medially to a level about 1 inch (2.5 cm.) below the pubic tubercle, i. e., well on to the scrotal swelling. The incision is then deepened to expose the external oblique aponeurosis in the upper and lateral part of the wound and the sac in the lower and medial part of the wound. A nick is now made in the external oblique aponeurosis, proximal to the external abdominal ring. The sac is then carefully lifted up, i. e., pinched up with two pairs of tooth forceps and incised between the two. The incision is then lengthened and the fluid in the sac allowed to drain out. The strangulated loops of intestine are then drawn out of the sac into the wound. These are now held by an assistant and covered by a pack with hot isotonic solution of sodium chloride. After these are thus secured, and only after, the incision in the external oblique aponeurosis is extended and the constriction divided. Such a procedure is adopted to avoid the possibility of the intestine's slipping into the abdominal cavity on sudden release of the constriction. The intestinal loops are now pulled out to examine the site of constriction on the bowel wall and the condition of the intra-abdominal portion of the intestines for a few inches beyond the site of strangulation. The necessary manipulation is then done and the loops returned into the abdominal cavity. The sac is now dissected bluntly up to its neck and ligatured at this point with a transfixion suture of surgical gut. The portion of the sac distal to the ligature is excised. No repair of the canal is done, and tightening of the internal abdominal ring is not attempted. The external oblique aponeurosis is now sutured with a continuous surgical gut suture and the skin with interrupted silk or silkworm gut sutures. In the postoperative treatment, stress is laid on giving the patient plenty of fluids. In addition to what he takes by

the mouth, he is given at least 2 pints (1.1 liter) of isotonic solution of sodium chloride or 5 per cent dextrose in isotonic solution of sodium chloride by the rectum every day for three consecutive days.

SUMMARY

1. Changes that take place in the mesentery, the blood vessels and the bowel wall are described.

2. From the observations based on these changes, it is suggested that there are probably seven stages in the evolution of a strangulated inguinal hernia. These stages are illustrated.

3. The stages in the formation of a hematoma in the mesentery and the probable sequence of changes consequent on its formation are suggested.

4. It is suggested that on the operating table these cases may profitably be divided into three groups—the first two including cases of the viable intestine and the third group including cases of the nonviable intestine.

5. An analysis of my 50 cases is made, all the important and interesting features being noted.

6. A brief description of the operation as routinely adopted by me is given.

The surgeons of the Sir Jamsetjee Jejeebhoy Hospital allowed me to operate on their patients with strangulated hernia. The superintendent of the Sir Jamsetjee Jejeebhoy group of hospitals allowed me access to the case records and permitted me to publish the cases. Mr. S. Deshaprabhu gave advice in the preparation of the illustrations.

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GASTRIC ULCERS PRODUCED EXPERIMENTALLY BY VASCULAR LIGATION

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VIRCHOW'S hypothesis [1] that human gastric ulcers were vascular in origin led to several attempts to reproduce similar lesions in animals by ligation of the gastric vessels. For the most part no significant results were obtained. Dogs were generally used, and short of total interference with the blood supply ligation had no effect on the intactness of the stomach.[2] This was largely due to a highly efficient collateral circulation. There is a complete circle of anastomoses around the two curvatures of the canine stomach,[3] and when the main arteries are tied the blood flow through intercommunicating branches is sufficient to maintain the nutrition of the organ. In human beings [4] the anatomic arrangement of the blood supply of the stomach is practically the same as that in dogs. Recently, Somervell [5] tied the gastric arteries in a

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large series of cases of duodenal ulcer, and no complications referable to the operation developed. A few experiments with ligation in rabbits were reported by Omata [6] and Graulich,[2e] with conflicting results. Since in all the species used previously the main gastric vessels were directly continuous with one another and thus provided the stomach with a rich collateral circulation, it was deemed worth while to observe the effects of ligation on the rat's stomach, which has a different type of blood supply.[7]

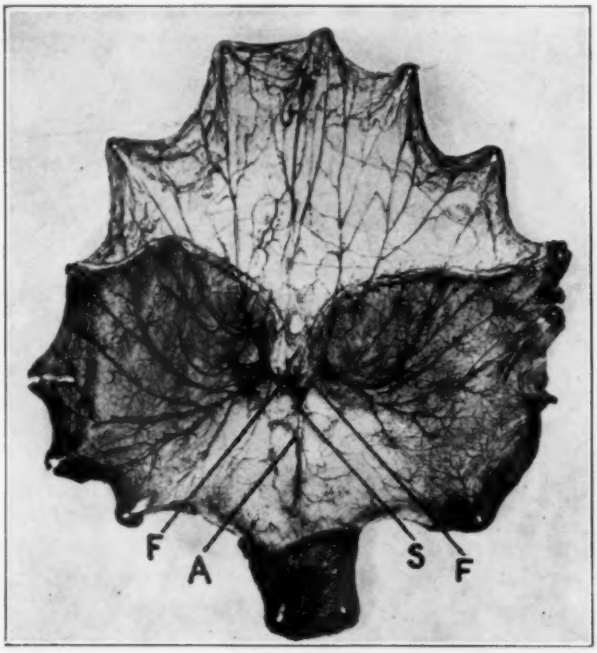

Fig. 1.—Injected and cleared normal specimen showing the blood supply of the rat's stomach. The three main branches arise from a common stem at *S* and are ligated close to their origin. The two fundic branches (*FF*) are longer and wider than the single antral vessel (*A*), which is barely visible. Arteries and arterioles are accompanied with veins and venules. The fundus is more vascular than the antrum or the rumen. × 2.

BLOOD SUPPLY OF THE RAT'S STOMACH

The rat's stomach [8] is divided by a transverse ridge into two parts, a nonsecretory rumen, or forestomach, and a glandular region, that is subdivided into an acid and enzyme–secreting fundus and a mucus-

6. Omata, T.: Experimentelle Studien über die Entstehung des runden Magengeschwurs, Virchows Arch. f. path. Anat. **269**:797-802, 1928.

7. Berg, B. N.: Vascular Changes in the Mucosa in Experimental Nutritional Gastritis, Gastroenterology **7**:340-354, 1946.

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secreting antrum (fig. 4). The main blood supply is derived from an artery which divides just below the junction of the esophagus with the stomach into three branches (fig. 1). Two of the vessels run on opposite sides of the organ, giving off several long branches to the fundus and rumen and a few short ones to the antrum. The third, shorter and thinner than the others, runs along the lesser curvature, sending small dichotomous twigs to the antrum and ending a little above the pylorus. The greater curvature receives short branches from a vessel in the

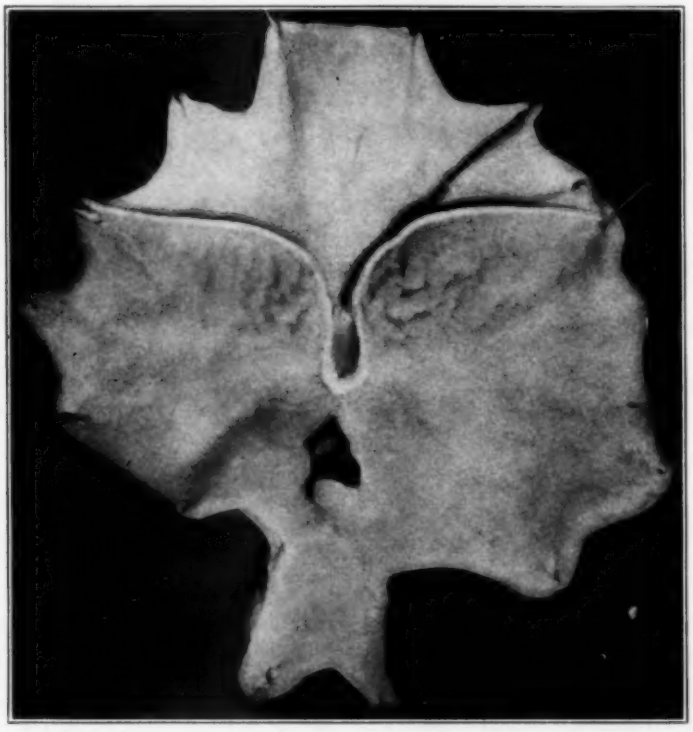

Fig. 2.—Antral ulcer twenty-four hours after ligation of the antral vessels. The stomach is opened along the greater curvature and is fixed in a 4 per cent solution of formaldehyde. × 2.

omentum which continues upward from the duodenum. Arteries and arterioles are accompanied with veins and venules.

Direct communication between the main gastric vessels, characteristic of human beings and dogs, is absent in rats. In the latter, the vessels terminate separately and connections between them are made through small branches in the submucosa and the capillary networks in the different layers of the wall of the stomach. The capillary plexuses in the mucosa of the fundus are more abundant than those in the mucosa of

the antrum or rumen and contribute to the greater vascularity of this part of the stomach.[7]

The observations herein reported are limited to the antrum and fundus; there is no counterpart of the rumen in the human stomach.

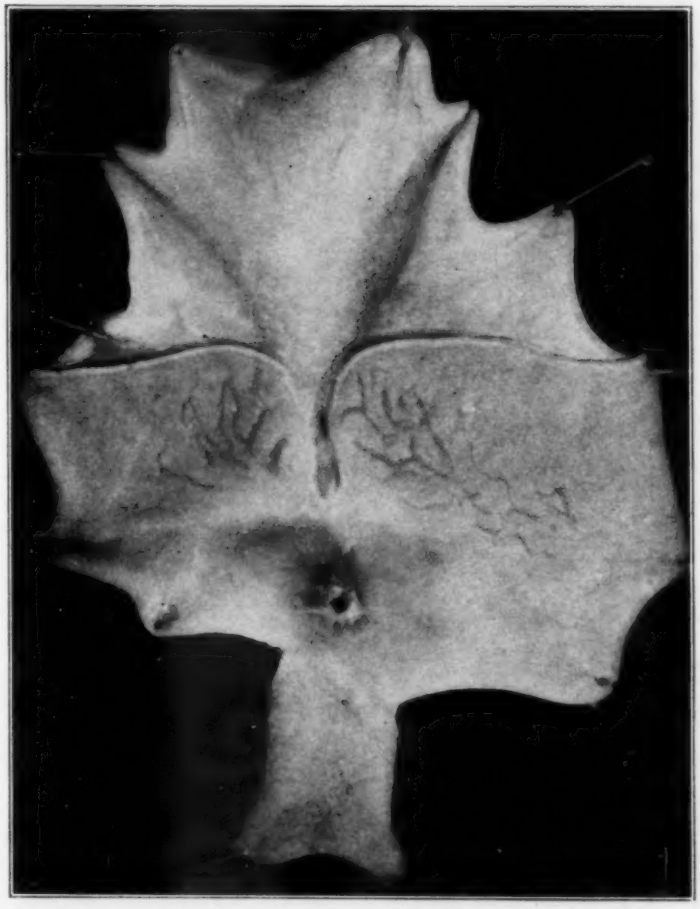

Fig. 3.—Healing antral ulcer sixteen days after ligation. A zone of hyperemia surrounds the remaining defect. Fixation was done in a 4 per cent solution of formaldehyde. × 2.

TECHNIC

Fifty-four albino rats of both sexes, ranging in age from 85 to 417 days, were used, and three types of experiments were performed. In group 1 the single artery supplying the antrum was tied; in group 2 the blood supply of one half of the fundus was interrupted, and in group 3 bilateral ligation of the fundic vessels was done. In each instance, the accompanying vein was included with the artery and the vessels were cut between ligatures close to their origin from the main stem. All the operations were performed with the animals under ether

anesthesia, and the stomach was mobilized through an incision in the upper midline. The animals were killed with ether twenty to twenty-four hours after ligation of the antral vessels and twenty-four or forty-eight hours after the fundic blood supply was cut off. The stomach was removed immediately, opened along the greater curvature and stretched with pins on a piece of cardboard. After being washed in isotonic solution of sodium chloride, the specimen was fixed in a 4 per cent solution of formaldehyde. Several rats in groups 1 and 2 were

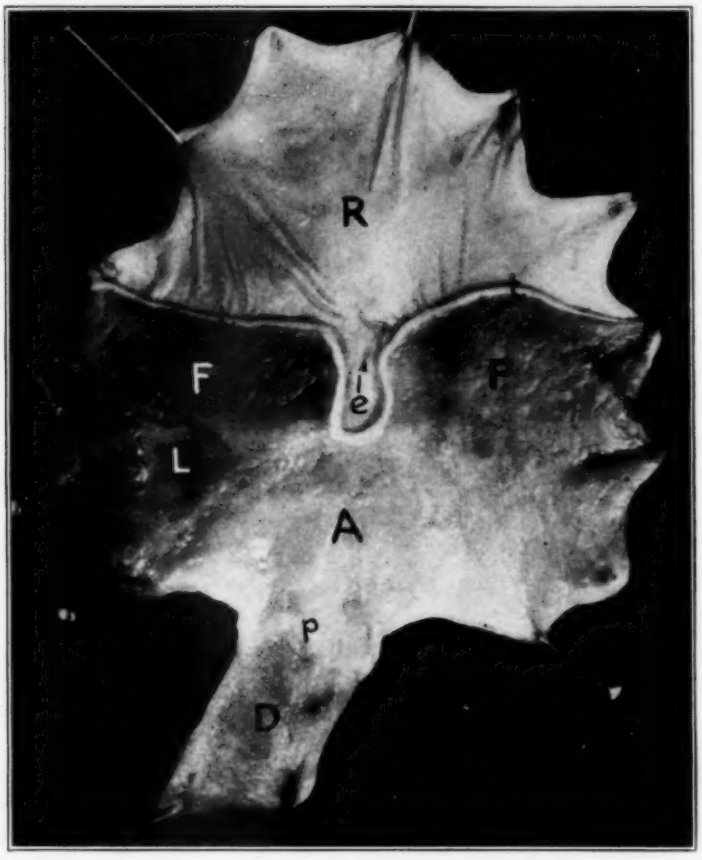

Fig. 4.—Fundic ulcer (*L*) twenty-four hours after unilateral ligation of fundic vessels. The differentiation of the glandular area into a fundus (*FF*) and antrum (*A*), which becomes indistinct after fixation, is shown clearly in this fresh specimen. The rumen, or forestomach (*R*), lies above the transverse ridge (*tt*), and the esophageal opening is at (*e*). The duodenum (*D*) begins at the pylorus (*p*). × 2.

examined eight days and sixteen days respectively after operation. In 9 animals, the gastric vessels were injected with india ink and the stomach was cleared in methyl salicylate after passage through graded alcohols according to a technic described elsewhere.[7]

RESULTS

Group 1.—Ligation of the Antral Blood Supply: The wall of the antrum was pale and edematous, and the swollen surface was devoid of its usual covering of mucus. In 10 out of 18 rats there was a single, punched-out, penetrating ulcer about 5 mm. in diameter situated in the center of the antrum (fig. 2). The defects had rounded margins and were oval, triangular, stellate or semilunar in shape. In several instances

Fig. 5.—Symmetric, sharply defined infarcts in the fundus twenty-four hours following bilateral ligation of the fundic vessels. The necrotic mucosa is still in situ, and after it separates an ulcer remains. × 2.

only the serosa remained in the base, and perforations were found twice. Blood clots were often present in the craters. Multiple small, irregular, superficial ulcerations of the mucosa were observed in 5 animals, and in 3 no grossly visible defects were noted.

Two rats examined after eight days had ulcers showing signs of repair. In 1 the ulcer was reduced to a small defect surrounded by a zone of intense hyperemia (fig. 3); in the other, there was still a con-

siderable crater but the margins were puckered. Of 2 animals after sixteen days there was a minute, irregular, superficial defect in 1 and a rather large, shallow lesion in the other.

Group 2.—Unilateral Ligation of the Fundic Vessels: In contrast with the pallor of the antral mucosa after ligation, the surface of the fundus was intensely congested and had a deep, purplish red color. In this series 12 rats were examined after twenty-four hours and 8 after

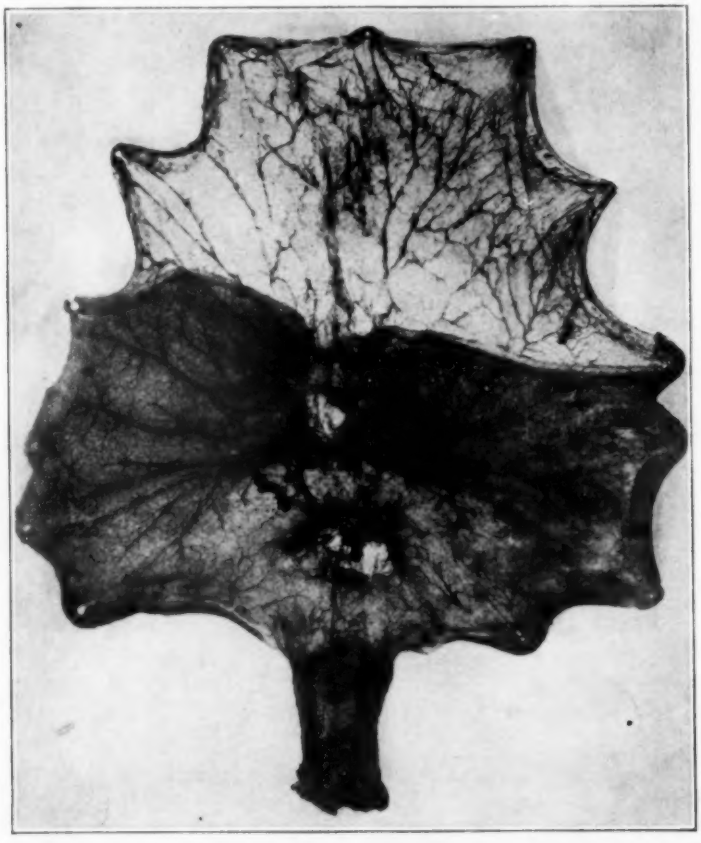

Fig. 6.—Antral ulcer surrounded by collateral vessels in an injected and cleared specimen. Compare with the less vascular normal antrum in figure 1. × 2.

forty-eight hours. In only 1 animal did there develop an ulcer, which consisted in a large, shallow, triangular-shaped defect (fig. 4).

Group 3.—Bilateral Ligation of the Fundic Vessels: The mucosa had a mottled appearance consisting of purplish red and reddish brown blotches. Five out of 8 animals killed after twenty-four hours had multiple ulcers in both halves of the fundus. The lesions varied considerably in size and shape; irregular deep fissures or larger stellate

defects were usually present, but in one experiment there were two extensive infarcts with necrotic mucosa still in situ (fig. 5). The latter finding was a good example of the changes antecedent to ulcer formation. After separation of the slough the remaining defect constitutes an ulcer . None of the lesions were perforated. In 2 rats multiple, minute, superficial healing ulcerations were found after eight days, and in 2 others the mucosa was normal after sixteen days.

COMMENT

The frequent occurrence of gastric lesions after vascular ligation in rats is in striking contrast with the normal results obtained in other species. This is probably due in large part to anatomic differences in the blood supply of the stomach. In rats, the main gastric vessels arise from a common stem and do not communicate directly with one another as they do in dogs and in human beings; instead they end separately, and anastomoses are made through terminal branches. The central part of the antrum is supplied by a single artery and vein, and when these vessels are tied a collateral circulation must be established through long, narrow anastomotic channels which are distant from secondary sources of blood supply (fig. 6). The fact that centrally located ulcers develop in a large number of animals shows that the collateral circulation is inadequate to keep the antral wall intact. The periphery of the antrum is preserved, however, by short branches from adjacent vessels along the fundic border and the greater curvature.

Since the fundus is more vascular than the antrum and has a better collateral circulation, it is not surprising that fundic ulceration is uncommon after unilateral ligation. Injected specimens show that an adequate flow of blood is provided through short anastomoses with branches of the intact fundic vessels of the opposite side, with branches of the vessels along the greater curvature and with antral twigs. When bilateral ligation is performed, however, the fundus loses most of its blood supply and ulcers appear. Collateral channels are reduced to intercommunications with vessels along the greater curvature and with antral branches, and these are insufficient to maintain the nutrition of the tissues.

It is noteworthy that despite extensive gastric ulcerations all the rats kept for periods of eight days or sixteen days survived ligation. Various stages of repair were observed, but healing was more advanced in fundic lesions than in antral lesions. The difference in rate of healing was probably attributable to the more rapid establishment of a collateral circulation in the fundus than of that in the antrum.

For many years the term "peptic" has been applied to ulcerations of the stomach in human beings. This connotation implies that peptic enzymes can digest living gastric mucosa, but there is no clinical or

experimental evidence to warrant this assumption. Certainly there is nothing in the present experiments on ligation to support the idea that enzyme action is related to the formation of ulcers. The experimental lesions are simply infarcts (fig. 5), and the size and depth of the defects left after separation of the dead tissue are determined by the effectiveness of the collateral circulation.

Previous studies [9] have shown that mucosal changes similar to those found in human antral gastritis develop in rats on a low calcium diet. However, further experiments [10] designed to induce deeper penetration of the antral wall by prolongation of the period of calcium deficiency have been unsuccessful; the defects become larger and the surrounding epithelial hyperplasia becomes greater, but the craters do not extend beyond the muscularis mucosae. These findings seem to indicate that a separate factor is concerned with involvement of the muscular coats after the initial break in the mucosa has taken place. Another possibility is that penetrating lesions are not preceded by a mucosal defect but are caused by a single factor which simultaneously affects all the layers of the gastric wall. An example of such a mechanism is provided by the experiments on ligation herein reported.

CONCLUSION

Owing to the anatomic arrangement of the blood supply of the rat's stomach, it is possible in this species to produce ulcers in the antrum and in the fundus by ligation separately of the vessels supplying these areas.

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9. Zucker, T. F.; Berg, B. N., and Zucker, L. M.: Nutritional Effects on the Gastric Mucosa of the Rat: I. Lesions of the Antrum, J. Nutrition **30**:301-317, 1945.

10. Zucker, T. F., and Berg, B. N.: The Time Factor in the Production of Gastric Lesions on Low Calcium Diets, Proc. Soc. Exper. Biol. & Med. **57**:1-3, 1944.

TUBERCULOUS DACTYLITIS IN THE ADULT

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TUBERCULOUS dactylitis in the adult is generally uncommon and its appearance rather rare. Although the practicing orthopedist and the busy pathologist can recall having seen a case or two of the condition in the adult, the literature, especially in North America, contains only scattered reports of cases. Furthermore, our conception of this lesion as described in the textbooks and more important works on tuberculosis is that of tuberculous dactylitis in childhood.

Tuberculous dactylitis or spina ventosa is usually a disease of infancy and childhood. Herzfeld and Tod [1] found 97 cases of the condition in 1,403 tuberculous children, thus representing 6 per cent of all cases of surgical tuberculosis. Sorrel and Sorrel-Dejerine,[2] in a larger sampling, reported an incidence of 14 per cent. In both series the disease became increasingly uncommon after 5 and scarce after 10 years of age.

No statistics on the incidence of adult spina ventosa were found. Stenström,[3] of Sweden, in 1935 reported 3 cases of tuberculosis in the phalanges of adults, for the purpose of describing differences between spina ventosa in the child and that in the adult. Isolated reports of cases from Germany,[4] France,[5] England [6] and Italy [7]

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* Fellow of the Dazian Foundation for Medical Research.

From the Orthopedic Services of Dr. Robert K. Lippmann, Mount Sinai Hospital, and Dr. Milton J. Wilson, Metropolitan Hospital.

1. Herzfeld, G., and Tod, M. C.: Tuberculous Dactylitis in Infancy, Arch. Dis. Childhood **1**:295, 1926.

2. Sorrel, E., and Sorrel-Dejerine: Tuberculose osseuse et osteo-articulaire, Paris, Masson & Cie, 1932, pp. 2-21.

3. Stenström, B.: Phalangeal Tuberculosis in Older Persons, Acta radiol. **16**:471, 1935.

4. Oehlecker, cited by Stenström.[3] Schinz, cited by Stenström.[3]

(Footnotes continued on next page)

imply the infrequent appearance of the lesion. In Christopher's textbook on "Minor Surgery"[8] there is an excellent photograph of the condition, reproduced through the courtesy of Dr. M. L. Mason, but no reference to a report by him.

The case presented herewith is of threefold interest. First, it demonstrates some of the differences between the lesion in the adult and that in the child. Second, it presents a problem in differential diagnosis. When the patient entered the Mount Sinai Hospital with an obvious pathologic fracture of the left index finger, he did not state the fact that he had been observed for several months in the Metropolitan Hospital Out-Patient Department, where many roentgenograms were taken. He did state, however, that he had been discharged from the Navy because a roentgenogram had shown a "tumor of the finger." Thus when roentgenograms were taken at Mount Sinai Hospital, because of the advanced stage of the disease, the lesion was interpreted as being the result of a tumor, syphilis or tuberculosis. A biopsy specimen suggested the latter. A few weeks later, after he had left the hospital, his past history was reinvestigated, and five old roentgenograms were found; thus the third interesting aspect, the fact that all the roentgenograms serially delineate the natural course of the disease from the initial to the final stage.

REPORT OF A CASE

History.—S. C., a 19 year old Negro factory worker, was admitted to the Metropolitan Hospital on March 30, 1941 with the history of a sudden attack of dizziness, weakness, pain in the right side of the chest and temperature up to 103 F. Physical signs and subsequent roentgenograms of the chest were indicative of a hydropneumothorax on the right side with partial collapse of the lung. In frequent aspirations large amounts of slightly turbid fluid were removed from the chest, which were on several occasions found to be positive for acid-fast bacilli. Inoculation tests on guinea pigs gave negative results however, as did also examinations of sputum and gastric aspiration. After a slow and prolonged course he was discharged in an improved condition on Dec. 22, 1941 and referred to the outpatient department for continued study.

During the next few months, while being observed in the outpatient department, a painless, progressive swelling of the proximal portion of the left index

5. Carpentier, C., and Hayem, A.: Tuberculous Osteoarthritis of the Index Finger in an Adult, Prat. méd. franç. **1**:1165, 1921-1922. Rieunau, G.: Tuberculosis of the Phalanges, Paris méd. **2**:287, 1933.

6. Heath, P. M.: Case of Tuberculous Dactylitis, Proc. Roy. Soc. Med. (Sect. Surg.) **14**:170, 1920-1921.

7. Antonioli, G. M.: Autoplastic Bone Graft in the Treatment of Spina Ventosa, Boll. e mem. Soc. piemontese di chir. **5**:1180, 1935.

8. Christopher, F.: Minor Surgery, ed. 4, Philadelphia, W. B. Saunders Company, 1940, p. 648.

finger was noted, and on May 23, 1942, in addition to the periodic roentgenograms of the chest, a roentgenogram was taken of the left hand (fig. 1), which revealed an elevation of the periosteum along the proximal half of the shaft of the proximal phalanx of the left index finger. There was indefinite cortical irregularity, especially at the radial aspect of the bone, and concomitant swelling of the surrounding soft tissue. Serologic studies and serial roentgenograms were suggested to determine the etiologic factor. In subsequent months of study reactions to frequent Wassermann tests were negative and periodic examinations of sputum gave normal results. Roentgenograms of the chest demonstrated no changes in the pulmonary fields.

On Sept. 18, 1943, because the patient began for the first time to experience pain in the left index finger, a second roentgenogram was taken (fig. 2).

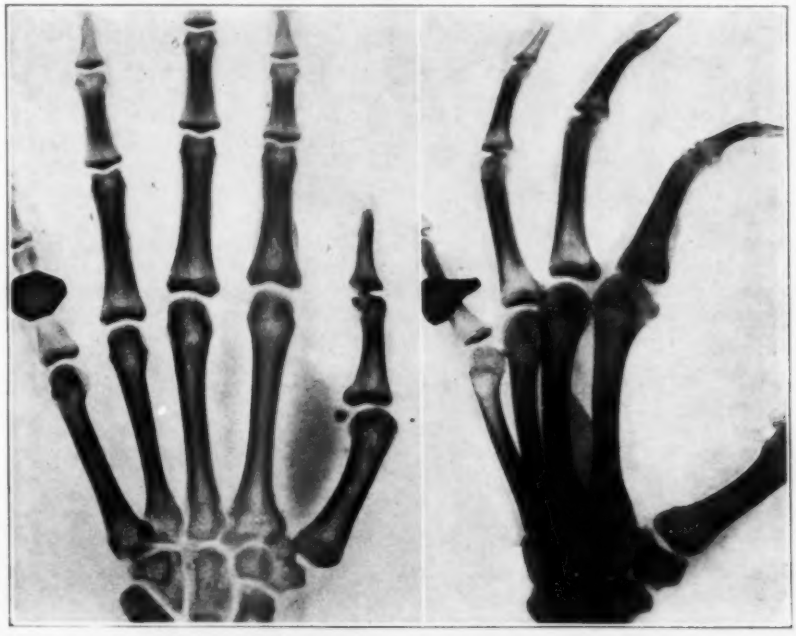

Fig. 1.—On May 23, 1942 there was elevation of the periosteum along the proximal half of the diaphysis of the proximal phalanx of the left index finger. The shaft was apparently uninvolved.

There was now revealed a large area of rarefaction involving the proximal half of the shaft, extending into the base and resulting in a moderate expansion of the bone. There was some sclerosis of the trabeculae in the distal half of the shaft, almost obliterating the cancellous portion of the bone but not extending into the head. The cortex of the volar surface of the involved portion of the phalanx appeared to be absent. A considerable increase in the swelling of the soft tissues had also taken place.

Two weeks after his last visit to the outpatient department he injured his left hand, and he was admitted to the Metropolitan Hospital, for the second time, on Oct. 4, 1943. The left index finger presented a fusiform swelling at

its base, greatest toward the radial side. The swelling was hard and indolent and appeared to be fixed to the underlying bone. There was tenderness to palpation only over the dorsum of the finger. A roentgenogram (fig. 3) showed considerable thickening of the distal two thirds of the proximal phalanx, with distinct cortical irregularity throughout the involved area and sclerosis in the

Fig. 2.—On Sept. 18, 1943 a large area of rarefaction in the shaft had replaced the periostitis and had resulted in moderate expansion. The remainder of the shaft showed sclerosis of the trabeculae almost obliterating the cancellous portion of the bone. There was partial destruction of the cortex.

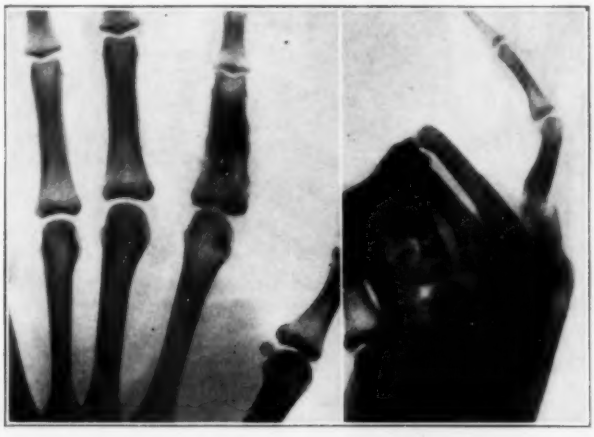

Fig. 3.—On Oct. 4, 1943 there was considerable thickening of the distal two thirds of shaft, with distinct cortical irregularity, and sclerosis had taken place. The proximal third showed irregular areas of decreased density with a pathologic fracture through it.

distal half of the shaft. The proximal third showed irregular areas of decreased density. An irregular transverse fracture was present through the center of the destructive area, which now extended into the base of the phalanx. Slight

ventral displacement of the distal fragment and anterior angulation at the site of fracture were noted. The swelling of the soft tissues had increased slightly. A punch biopsy of the lesion yielded insufficient diagnostic evidence, and the patient was discharged from the hospital.

He was seen again in the outpatient department on Nov. 23, 1943, where another roentgenogram was taken (fig. 4). This roentgenogram showed further destruction of the phalanx. The fracture was not healed, but the fragments were in good alinement. There was no change in the swelling of the soft tissues.

A few weeks later he entered the Navy, had roentgenograms of the chest and passed the physical examination. The swelling and disability, however, prevented full use of the finger, and he was discharged after one month. He returned

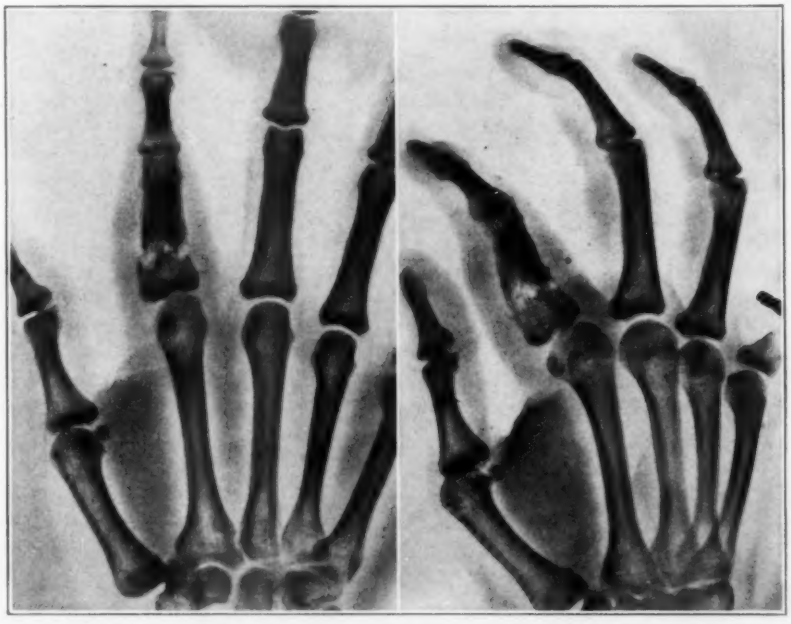

Fig. 4.—On Nov. 23, 1943 further destruction and loss of bone substance at the fracture line were noted. The ever present soft tissue swelling is shown clearly in this figure.

to the outpatient department for the last time on Feb. 1, 1944, where roentgenologic examination of the left hand (fig. 5) showed the previously noted destructive lesion of the proximal phalanx with widening of the shaft. There was formation of new bone in the involved portion of the shaft and base as well as bony callus at the dorsolateral aspect of the site of fracture and still considerable irregularity of the adjacent ends of the fracture where union had not occurred.

He was able to work in a factory and was asymptomatic until April 12, 1944, when he entered the Mount Sinai Hospital with a five day history of severe pain in the left index finger following a fight in which he struck his adversary on the head with his left hand.

Examination.—The patient was a well developed, muscular Negro, complaining of pain in his left index finger. Significant physical findings were confined to

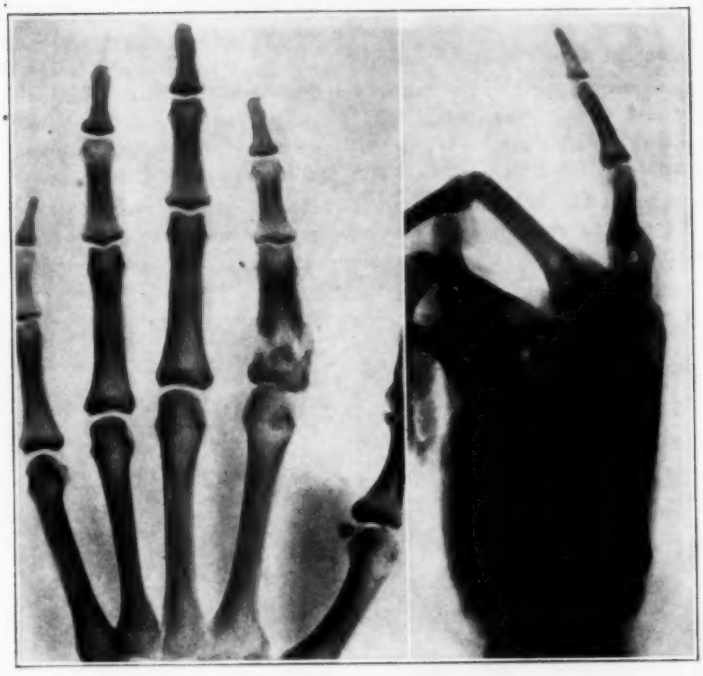

Fig. 5.—On Feb. 1, 1944 destruction of the phalanx was continuing, but there was some evidence of formation of new bone and callus.

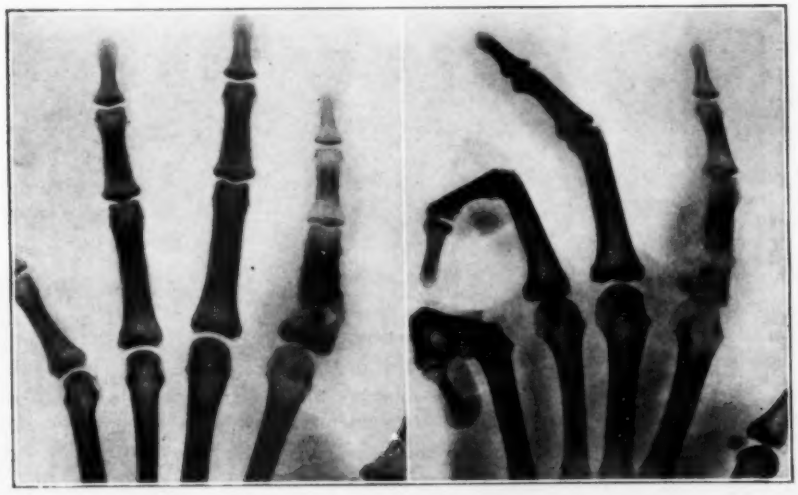

Fig. 6.—On April 12, 1944 there was increased extensive destruction with bony debris extending out into the soft tissues and a widened sclerotic shaft. The medullary cavity was almost completely obliterated.

the left hand, where the index finger presented a bony, hard, fusiform swelling of the proximal phalanx, greatest toward the radial side. Crepitus could be elicited over the midportion of the swelling, and there was moderate tenderness at this site.

Laboratory Data.—Examination of the blood revealed a hemoglobin content of 108 per cent, 10,600 leukocytes and a normal differential cell count. A Wassermann test elicited a negative reaction; the erythrocyte sedimentation time was normal. The urine showed a faint trace of albumin. Roentgenologic examination of the chest showed no abnormality in the lungs. Both costophrenic sinuses were obliterated by adhesions. Examination of the left hand showed a pathologic fracture through the base of the proximal phalanx of the index finger, at which site there was evidence of an extensive destructive process. Bony debris extended out into the soft tissues, which showed considerable swelling. The shaft was widened and sclerotic, and the medullary canal was almost completely obliterated (fig. 6).

A biopsy was performed, and a considerable amount of loose, ragged bone was encountered at the site of the fracture. This was curetted out, and much white tissue having the appearance of a tumor was obtained. Microscopic examination revealed a granulomatous and necrotizing osteitis with extensive necrosis, but no acid-fast bacilli were found.

Operation.—A racquet-shaped incision was made completely encircling the base of the left index finger and extending proximally on the dorsoradial surface of the second metacarpal for half its length. The superficial tissues were incised in a circumferential fashion. The extensor and flexor tendons were then transected and allowed to retract back. Next, the metacarpal was exposed and its head and neck resected obliquely from the first to the second web space to eliminate the protrusion of this portion of bone. The entire finger and head of the metacarpal were then removed. Flaps were mobilized and the skin closed.

Pathologic Report (Dr. Paul Klemperer).—The specimen was a left index finger resected through the metacarpal bone. The proximal part of the first phalanx and the adjacent tissue were replaced by a large amount of grayish, necrotic material, giving the digit a spindle shape, the typical appearance of a spina ventosa.

Histologic examination showed diffuse granulation tissue with scattered areas of coagulation necrosis, which infiltrated the periosteum and the adjacent soft tissues. It included many epithelioid cell tubercles with typical Langhans giant cells. The same process extended into the bone and between the often irregular bone trabeculae; frequent tubercles were seen and but little normal fat marrow, substituted for the most part by the tuberculous granulation tissue. The pathologic diagnosis was tuberculous osteomyelitis and periostitis and spina ventosa.

Postoperative Course.—The wound healed by primary union, and recovery was entirely uneventful.

Follow-Up.—For one year after operation the patient worked as a plater, without difficulty, and there was a good functional and cosmetic result. There are no systemic or further osseous manifestations of tuberculosis.

COMMENT

The various roentgenographic changes seen in tuberculous dactylitis are portrayed in many books and articles, and the subject is adequately

discussed, that is, for the childhood form. Sorrel and Sorrel-Dejerine [2] present serial roentgenograms depicting the evolution of this form. For the adult type, however, only casual references [9] to differences are made or a single roentgenogram is shown of one phase of the picture and taken to represent the entire pattern of change.

The earliest roentgenologic evidence of involvement of the bone in both the childhood and the adult form of the disease is elevation of the periosteum (fig. 1). This elevation, at first the result of an underlying serous effusion,[10] is indicated by a linear deposit of new bone surrounding part or all of the diaphysis. At this time or shortly after, changes in the cortex may be represented by an indefinite irregularity. Although at first there is usually no evident swelling of the soft tissues, this soon occurs and progressively increases as the disease process advances.

In the child the periosteal reaction is intense, and increase in thickness and expansion of the cortex result. This may be so distinct that the normal shaft may be seen within the expanded periosteal sheath of new bone.[9a] Gradually, however, destruction of the shaft occurs, and it separates as a sequestrum. The sequestrum shows progressive fragmentation, and much of the internal contents of the involucrum are absorbed, leaving a cystlike cavity which appears to be ballooned or injected with air, the so-called spina ventosa, and restoration of bone is gradual. Often, however, the sequestrums begin to suppurate, and within this pus the new periosteum necroses. At this stage fistulas exist, through which small fragments of the sequestrum are extruded. The disease may advance and destroy the neighboring joint.

Now these changes and the greater incidence in childhood, especially in the infant, may be explained in part by certain anatomic facts. Primary tuberculosis of the bone shows predilection for well vascularized bone. In the child there is a large artery in the diaphysis of the short tubular bones, which favors lodgment of infectious material, and these bones still contain spongy bone instead of a marrow cavity until the fourth or fifth year of life. Also, the periosteum has a

9. (*a*) Brailsford, J. F.: The Radiology of Bones and Joints, ed. 3, Baltimore, Williams & Wilkins Company, 1945, pp. 28-29 and 38-39. (*b*) Hodges, P. C.; Phemister, D. B., and Brunschwig, A.: The Roentgen-Ray Diagnosis of Diseases of Bones, New York, Thomas Nelson & Sons, 1938, p. 400. (*c*) Sorrel and Sorrel-Dejerine.[2]

10. Golding, F. C.: Tuberculosis of Bone, in Shanks, S. C.; Kerley, P., and Twining, E. W.: A Textbook of X-Ray Diagnosis, London, H. K. Lewis & Co., 1939, vol. 3, pp. 414-415.

regenerative power of reaction which is lacking in adult life.[2] In the adult the initial periostitis is replaced by a large area of rarefaction, and the finer cancellous structure and the compact cortex are absorbed, leaving a coarser cancellous model of the bone which shows a moderate expansion (fig. 2). The cortex may be partly eroded or destroyed, and there may be sclerosis of the trabeculae in the adjacent portion of the shaft with a tendency to obliteration of the cancellous bone. The cortical irregularity and sclerosis increase as the bone thickens, while the coarse, cancellous, irregularly destroyed area progresses and may appear honeycombed. At this stage pathologic fracture is most likely and appears to be somewhat common (fig. 3). With further destruction, delayed healing and continued trauma, there appears a decidedly rarefied zone, giving the appearance of loss of bone substance as in cases of nonunion (fig. 4). In a later stage callus formation occurs, and there are manifestations of formation of new bone (fig. 5). In the final phase there is, however, increased, extensive destruction of bone, and this debris may be seen extending out into the soft tissue (fig. 6). The remainder of the shaft is widened and sclerotic, and the medullary cavity is almost completely obliterated but there is no large sequestrum, no involucrum and no fistula formation. It may be that these last changes are inevitable, but in this case and in those reported ablation of the finger precluded their appearance.

Since tuberculous dactylitis in the adult is rarely seen, diagnostic criteria without biopsy are difficult to establish and several simulating lesions require differentiation. Syphilitic dactylitis is probably the most difficult lesion to be excluded. Here, however, more than one finger may be involved. Roentgenograms show distinct periostitis, and the bone is increased in breadth principally by the deposition of subperiosteal new bone with but little influence from expansion. There is greater density to the bone and less evidence of necrosis. Evidence of syphilis elsewhere (especially periostitis of the long bones) and response to antisyphilitic therapy are conclusive.

The phalanges are a favorite site for the occurrence of enchondromas. The central type of solitary enchondroma may be a problem when there is present expansion of the phalanx consisting in a translucent area with trabeculation surrounded by a sclerotic bony capsule.[10] But there is no periostitis and the cortex is attenuated. The tissue is generally devoid of calcium but contains irregular nuclei or islands of calcium throughout, which are indicative of the chondromatous nature of the tumor.[9a]

The development of osteitis in the phalanx of a young adult, in the absence of obvious infectious or traumatic causation, with a woolly out-

line of reacting periosteum [9a] or the striking destruction and fragmentation of bone as seen in the end stage of tuberculous dactylitis may give rise to the suspicion of sarcoma. The localization and monostosis of the former and metastasizing feature of the latter are valuable diagnostic aids.

The lesions of the bone in Boeck's sarcoid are not always punched out and cystic in nature.[11] It has been pointed out [12] that these lesions may begin merely as a coarsening of the trabecular pattern, after which areas of both cortical and central destruction appear, and as the disease progresses the destruction may be extensive. This pattern is exceptional, however, and typically one sees a multiplicity of small cystic areas situated centrally in the ends of the phalanges.

In coccidioidomycosis bony lesions are usually multiple, involve cancellous bone and often are associated with destruction of bone and formation of new bone, but the solitary lesion is practically indistinguishable from tuberculosis.[13] In fact, giant cells and caseation occur, and only the finding of the double-contoured spherules makes the diagnosis. The report of a case of the condition in a phalanx is illustrative.[14] The history of infection in an endemic area and positive reactions to a coccidioidin test of the skin and complement fixation test are highly suggestive of coccidioidomycosis.

Leprosy and yaws in nonendemic areas may be mistaken for tuberculous and syphilitic dactylitis. Predominant cancellous dissolution and difference in localization in giant cell tumors and solitary bone cysts are of differential diagnostic value. Osteomyelitis of a phalanx would be most unusual and might be accompanied with clinical signs of sepsis, sequestration and later pyogenic arthritis.

Aside from the roentgenographic differences between the childhood and adult forms of tuberculous dactylitis, others are manifest. The frequency of fistula formation in the child contrasts sharply with its absence in the adult and has already been mentioned. As in this case, 1 of the 3 patients in the cases Stenström [3] reported displayed a pathologic fracture. Pathologic fractures in tuberculosis of the bones and joints are unusual. This is perhaps because in advanced tuberculosis of the long bones with involvement of the adjacent joint either the patient is

11. Holt, J. F., and Hodges, F. J.: Significant Skeletal Irregularities of the Hands, Radiology **44**:23, 1945.

12. Doub, H. P., and Menagh, F. R.: Bone Lesions in Sarcoid: A Roentgen and Clinical Study, Am. J. Roentgenol. **21**:149, 1929.

13. Carter, R. A.: Infectious Granulomas of Bones and Joints, with Special Reference to Coccidioidal Granuloma, Radiology **23**:1-16, 1934. Smith, C. E.: Parallelism of Coccidioidal and Tuberculous Infections, ibid. **38**:643, 1942.

14. Goren, M. L.: Localized Coccidioidomycosis of Bone: Report of a Case, J. Bone & Joint Surg. **28**:157, 1946.

bedridden or the part is protected by a cast or appliance. In tuberculous dactylitis of childhood these fractures are not seen, probably because there is much formation of new bone. In fact, with the exception of tuberculous dactylitis (and tuberculosis of the metacarpals and metatarsals), this degree of periosteal reaction and thickening of the bone is not observed in osseous tuberculosis.[15] In the adult form, on the other hand, the predominant feature is osteitis with destruction, and, with easy accessibility of the part to trauma, fracture of the bone is enhanced. In some of the cases seen by Kohlmann [16] there was so much bone destruction that the phalanges were broken up into fragments.

The tuberculous child is usually hospitalized because of pulmonary lesions or generalized tuberculosis, and thus dactylitis frequently is an incidental finding of a generalized process. By the same token, multiple lesions are not uncommon. Contrariwise, in the adult dactylitis is not part of a generalized process, and multiple lesions are usually not found.

Clinically, there are no reports of shortening or contracture of the involved finger in the adult. This deformity, however, is often present in the child, especially during the healing stage.[17] Two factors may be responsible for this change: first, disturbance in growth resulting from destruction of the epiphysial plate of the involved phalanx and, second, the pull of the tendons of the finger imposed on the contracting granulation tissue.[18]

Finally, the treatment of the two forms of dactylitis appears to be different. It is felt that the outlook in the childhood type is good and that with the usual immobilization treatment and general antituberculous therapy many lesions will spontaneously heal.[19] In cases in which operation has been indicated, good results have been reported with subperiosteal resection and curettage [20] followed in some cases by bone grafts.[21] In the stage of sequestration, sequestrectomy is the

15. Webster, R.: Tuberculous Dactylitis, M. J. Australia **2**:55, 1938.

16. Kohlmann, cited by Stenström.[3]

17. Mercer, W.: Orthopaedic Surgery, ed. 3, Baltimore, Williams & Wilkins Company, 1943, pp. 299-301.

18. Kaufmann, E.: Pathology, translated by S. P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 2.

19. Dalto, A.: Spina Ventosa Cured in an 18 Months Old Child, Prensa méd. argent. **42**:2012, 1941. Nesti, D.: Conservative Treatment of Tuberculosis of the Bone, Policlinico (sez. prat.) **40**:431, 1933. Mercer.[17]

20. Roger, J. P.: A Case of Spina Ventosa, Bull. Soc. med. d. hôp. Univ. de Quebec **35**:181, 1934. Herzfeld and Tod.[1]

21. Fontaine, R.: Double Spina Ventosa, Metacarpal and Metatarsal, Treated by Extirpation of the Lesions and Resection of the Diaphysis Followed by Osseous Grafts, Rev. de chir., Paris **75**:639, 1937. Antonioli.[7]

method of choice.[2] In the adult form of dactylitis the therapeutic armamentarium is limited. It might be supposed, in the absence of precedent, that immobilization of the finger and general measures are worthy of trial. However, tuberculosis of bone in which the disease involves or threatens the adjacent joint in the adult is, in general, regarded as more dangerous than that in the child, and immobilization becomes synonymous with fusion. Now, since the finger does not lend itself easily to such a procedure, eradication by means of ablation becomes a definitive step. Furthermore, the liability of recurrent fracture, with its consequent impairment of function of the finger and the decidedly destructive nature of the lesion, makes amputation proximal to the metacarpophalangeal joint a somewhat beneficent eventuality.

METHOD OF CORRECTION OF ECTOPIA CORDIS

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DEFECTS of the sternum are not common. They may vary from simple notching of the manubrium and irregularities in shape of the xiphoid to complete absence of the entire structure.

The minor defects are not usually recognized and are discovered incidental to a roentgenogram of the chest. The larger defects are brought to attention at birth, because of their effects on the thoracic and mediastinal contents. These effects may be varied, depending on the extent of the defect. Herniation of the lungs will cause decided disturbances of respiration. Lack of normal restraint of the heart and associated large vessels may cause profound circulatory phenomena. This last type of defect attracted my interest, and in studying the literature I was impressed by two facts, namely, (1) the high mortality rate and (2) the attitude of hopelessness on the part of surgical attendants in discussing such cases.

With these facts in mind, an intensive study was made in an effort to correct some of these defects. This study led my associates and me to believe that most of these patients could be benefited if they were properly prepared and the operation done in stages. I should like to report 2 cases of ectopia cordis and offer a method that I used in both cases.

REPORT OF CASES

CASE 1.—J. S., a white boy aged 7 weeks, entered the hospital on Jan. 14, 1943. The chief complaint was paroxysmal attacks or rapid heart beat accompanied with cyanosis.

Physical Examination.—The head and scalp were normal. There was bulging of the anterior surface of the neck between the two sternocleidomastoid muscles. This became larger at times. In the chest anteriorly, the ribs did not meet in the midline, and there was an absence of the entire manubrium and the body of the sternum. Only a thin layer of skin lay over the defect, and with each pulsation of the heart the cardiac structures raised the skin above the level of the thoracic wall. When this act took place, the large vessels leading from the heart could be seen. The lower end of the sternum showed the xiphoid present. The abdomen was normal.

The probable diagnosis was a congenital defect of the sternum with partial ectopia cordis.

The child was placed under an oxygen tent temporarily, and during the next week a formula was supervised, with definite improvement of the child. During

the second week the oxygen tent was removed, but the child continued to have paroxysms of cyanosis and difficulty in breathing.

Roentgenograms were made showing the defect. Consultants of the general surgical service were of the opinion that no surgical therapy was indicated. Because of the unusual type of case, it was decided that the child would not get better and that it was worth trying some procedure in an endeavor to relieve the acute situation.

On Feb. 11, 1943, with the patient under nitrous oxide and ether anesthetic, an incision was made on the right side of the lower part of the chest. A block of

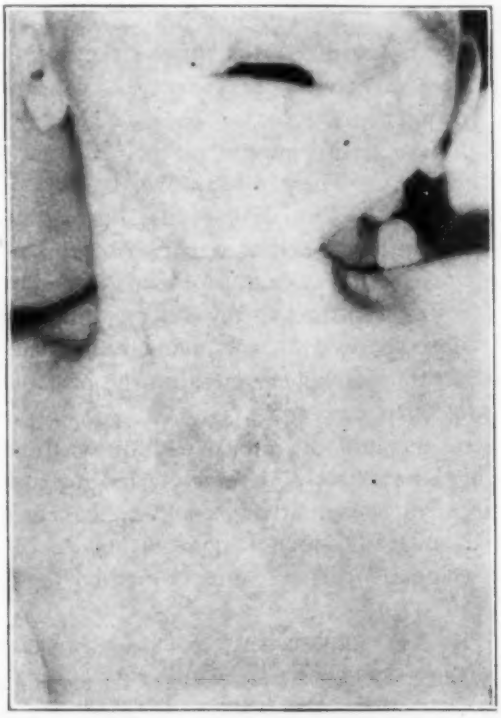

Fig. 1 (case 1).—Patient aged 7 weeks. Anterior view, showing defect of the sternum.

cartilage which included the eighth and ninth costal cartilages, measuring 1½ inches (3.8 cm.) in diameter, was removed en masse. About twenty-five minutes was required for this procedure. The child's pulse became somewhat irregular. It was decided to close the wound. The cartilage was placed in plasma and refrigerated. The wound healed by primary union.

On Feb. 18, 1943, with the patient again under anesthesia induced with nitrous oxide and ether, the anterior part of the chest was prepared, and, with Dr. Rix holding the heart in the thoracic cavity by direct pressure, a 4 inch (10 cm.) transverse incision was made across the midportion of the hiatus. By sharp dissection, the cardiac structures were dissected loose from their attachment to the skin and surrounding cervical muscles. There was no definite pericardium. This permitted the heart to descend into the thoracic cavity. The soft structures

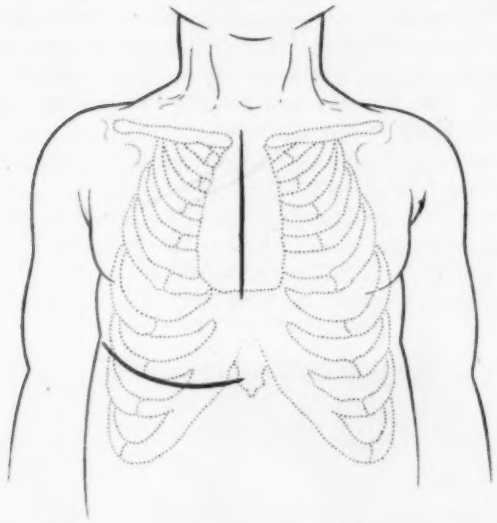

Fig. 2.—Diagram outlining location and types of incision.

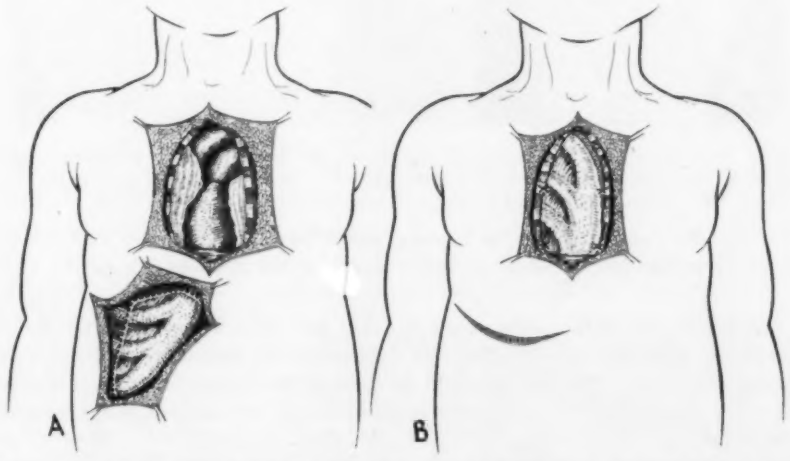

Fig. 3.—*A*, diagram showing exposure of the sternal defect and outlining of the massive graft of cartilage to be removed. *B*, diagram showing graft wired in position.

were then mobilized. The piece of cartilage previously removed and preserved in the plasma was laid across the hiatus, and two silver wire sutures were placed through the costal cartilages, pulling the lateral sides of the opening medially and also serving to anchor the cartilage graft. The muscles were then pulled over about halfway on each side. These were sutured with interrupted black silk sutures. The skin and subcutaneous tissues were closed with buried silk and interrupted black silk in the skin.

The convalescence was satisfactory in that the paroxysmal attacks of cyanosis stopped and the pulse rate slowed. The healing was clean, with two exceptions. There were two small sinuses leading down to the wires; so on March 22, 1943, with the patient under nitrous oxide and ether anesthesia, the wires were removed. The wounds were filled with sulfanilamide powder. Healing was prompt, and the child was discharged on April 16, 1943, completely healed and gaining weight.

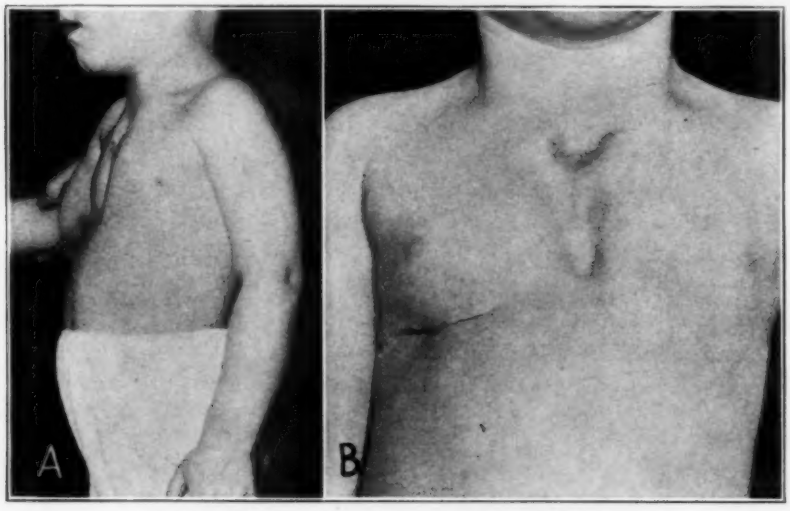

Fig. 4 (case 1).—Patient aged 3 years. *A*, lateral view, showing repair. Attention is called to freedom of breathing. *B*, anterior view, showing repair.

His weight on entrance to the hospital was 9 pounds, 7¼ ounces (4,280 Gm.). His weight when discharged from the hospital was 12 pounds, 3½ ounces (5,532 Gm.).

Follow-up Observation.—On April 25, 1943 the child was gaining weight and progressing satisfactorily. On Dec. 10, 1943, growth was normal and the child's condition was good. On Jan. 14, 1946, at the age of 3 years, the patient weighed 30 pounds (13.6 Kg.); he was well developed and had no symptoms referable to the chest.

Case 2.—This case had been referred to me seven years before by Dr. William Eastland. My records of May 8, 1937, show that the patient had a defect of the anterior part of the sternum and manubrium which measured 2 inches (5 cm.) in diameter. This defect was covered with a thin, parchment-like skin, through which could be seen the pulsating heart. Surrounding the defect the bony structures were rather firm.

I was at a loss as to what to do for her but evolved the plan of having the mother make a snug 5 inch (12.7 cm.) thoracic binder with a 3 inch (7.6 cm.) pocket on the inside, in the portion of the binder that would cover the defect. Into this pocket was placed a piece of sponge rubber. When the binder was adjusted and made tight, the sponge rubber acted as a truss and held the heart within the thoracic cavity.

I saw the child several times during the next two years, and, although she was nervous, she was growing and the mother had made her new binders to fit as she grew. I then lost contact with her, but after the success in this later case I started a search for her.

When found, she had reached the age of 12 years and was thin and small for her age. She had continued to wear the type of support that we had fitted her with at the age of 5 years. Her condition was discussed with her parents, and they agreed to have an attempt made at correction.

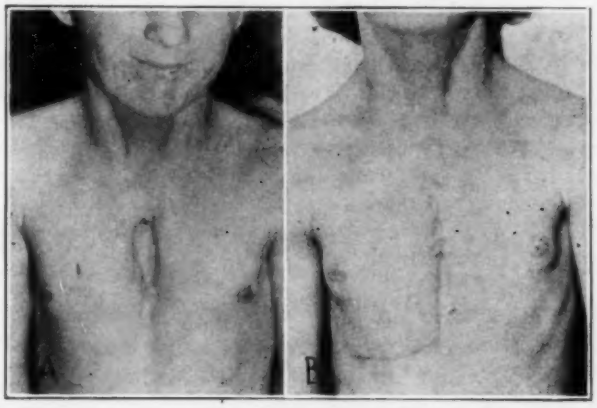

Fig. 5 (case 2).—*A*, patient aged 12 years. Anterior-posterior view, showing defect of the sternum. *B*, patient aged 13 years. Note the striking improvement in development and smooth repair of the thoracic wall.

On Sept. 21, 1944, she entered the Crippled Children's Hospital, and the following report is a history of her case.

B. J. H., aged 12 years, was an extremely nervous, pale thin girl. Her eyes were prominent. Over the chin and neck were many telangiectases from irradiation of an old hemiangioma.

Her neck was long and slender. The chest was symmetric. Anteriorly, there was a defect of the upper two thirds of the sternum which permitted the heart to make partial excursions out of the chest with each diastole. The heart rate was 84. The blood pressure was 90 systolic and 50 diastolic. There was some clubbing of the fingers. Nothing else abnormal was found in the physical examination.

The child was put to bed and given small doses of phenobarbital with frequent high calory feedings.

On Sept. 25, 1944, she was taken to surgery, and the following report gives the details of the procedure:

Observations.—There was a defect of the anterior part and upper two thirds of the sternum which permitted the heart to make excursions partially out of the chest with each diastole. The skin was thinned out and parchment-like over the area of this defect. The heart was clearly visible through the skin. When the skin was freed, it was found that the pericardium was only partially formed and that there was no pericardium over the anterior aspect of the heart.

Operation.—White soap, water and alcohol were used to prepare the anterior part of the chest. A 5 inch (12 cm.) transverse incision was made over the seventh, eighth and ninth right costal cartilages, and a portion of all these cartilages was removed, so that a block of cartilage measuring 3 inches (7.6 cm.) in diameter was removed. The wound was closed in layers by interrupted black silk.

A midline vertical incision was made from the level of the sternoclavicular articulation down to the level of what would normally be the level of the xiphoid process. This measured 7 inches (17.7 cm.) in length. By sharp dissection, the pericardial sac was freed from its border attachments to skin. The fascia and muscular fibers were mobilized and brought together by interrupted silk sutures to close the defect in the pericardium. The entire wound was then dusted with sulfanilamide crystals. The cartilage graft was laid in place and anchored with wire sutures. The skin was closed with interrupted black silk. The patient's condition was good.

On Oct. 13, 1944 she was discharged from the hospital. At a follow-up examination on March 15, 1945, the wound was solidly healed. The child was leading a more active life, and the mother stated that she was decidedly less nervous.

On Sept. 23, 1945 she was in excellent condition. She had gained 5 pounds during the summer and was more active.

PROGRESS IN ORTHOPEDIC SURGERY FOR 1945

A Review Prepared by an Editorial Board of the American Academy of Orthopaedic Surgeons

I. DISEASES OF GROWING AND ADULT BONE

Prepared by

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Growth of Bone.—Hendryson [1] has written a stimulating article in evaluation of the method of estimating growth of long bones published by Digby in 1916. He attempts also to arrive at a standard mean of growth in a large series of cases. His report is based on measurements of fifty-four human femurs. Needles were passed into the nutrient foramens. Mensuration from the point where they crossed the center of the medullary cavity to the obliterated epiphysial line revealed an astonishing degree of variation. Instead of growth of the distal segment of 69 per cent, as found by Digby and generally thought to be constant, the distal femoral segment was found to vary decidedly, the greatest growth being 79 per cent and the smallest 50 per cent. The percentage growth mean for the series, however, was 71 per cent, which is surprisingly close to Digby's estimate. In projecting needles into the six femurs of the series with dual arterial supply, Hendryson states that the "expected intersection of the prolonged nutrient canals did not occur in 4 instances." It should be pointed out that Digby stated that they converge on "almost" the same point in the medullary cavity. The author concludes that Digby's method of determining percentage of growth from a given epiphysial line in a long bone is unreliable and without foundation and that the figures derived from the method are inaccurate. One cannot help feeling that the average percentages of Hendryson tend to confirm Digby's estimate and strengthen the clinical impression that the increment of growth at the distal femoral epiphysial plate is about 70 per cent. Hendryson has carried out Digby's suggestion that "a thorough examination of a large series of each bone is desirable to provide reliable averages." It is realized that epiphysiodesis in the equalization of leg length is not mathematically accurate, but it is generally considered an excellent procedure and the results in reported series of cases have been favorable. Hendryson's article will not change the method of determining expected growth of an extremity,

1. Hendryson, I. E.: An Evaluation of the Estimated Percentage of Growth from the Distal Epiphyseal Line, J. Bone & Joint Surg. **27**:208-210 (April) 1945.

but it is sure to precipitate reinvestigation of the question of epiphysial growth.

Kennedy [2] has studied the clinical histories of a series of children to determine in what conditions precocious skeletal development occurs and the factors which may be responsible for skeletal precocity. The histories of 23 patients are reviewed, and, on study, show that precocious skeletal development is not a peculiarity of any one syndrome. It may be found with or without obvious endocrine disorders and may follow the administration of endocrine preparations. Considerable material exists on retarded skeletal development. This article is an addition to knowledge of skeletal precocity.

Woolley and McCammon [3] have constructed a curve of radial growth during the first two years from measurements of 400 normal infants. They postulate that the development of osseous structure is dependent on thyroxin and that deficiency is manifested not only by failure of the center of ossification to develop but by striking decrease in linear growth and by the appearance of a roentgenologically visible linear density at the epiphysial cartilage. This dense line, they say, does not resemble that of rickets, scurvy, syphilis or lead poisoning. Adequate treatment in congenital cretins causes new bone of normal appearance to develop distal to the dense bone in three to four weeks, and as treatment continues growth parallels the normal curve. It is emphasized that the standard is applicable chiefly to congenital myxedema and only until 2 years of age. For some time the chronologic appearance of centers of ossification has been the most useful index of thyroid function during infancy. This is an interesting variation, and, since changes occur rapidly, it should be useful as a guide in the control of therapy in young cretins. The authors state that the administration of a therapeutic test with thyroid substance should be reflected visibly in the long bones within three to four weeks if true lack is present. If not, no changes will be observed.

Variation in growth within the epiphysis of the lower end of the femur is imputed as a possible cause of osteochondritis dissecans by Ribbing.[4] He postulates the presence during childhood of a superficial accessory nucleus of bone. He is of the opinion that the loose body develops from one of these nuclei which may be imagined to remain throughout childhood relatively isolated from the surrounding bone tissue by islands and bands of persistent epiphysial cartilage, the vascular supply

2. Kennedy, R. L. J.: Precocious Skeletal Development, J. A. M. A. **127:** 580-581 (March 10) 1945.

3. Woolley, P. V., Jr., and McCammon, R. W.: Bone Growth in Congenital Myxedema, J. Pediat. **27:**229-235 (Sept.) 1945.

4. Ribbing, S.: Zur Ätiologie der Osteochondrosis dissecans, Acta radiol. **25:**732-755, 1944.

of which is only incompletely connected with the neighboring vessels by colateral circulation. He states the belief that a bony nucleus which is only partially connected with its surroundings might well become detached by minor traumas even though the articular cartilage remained intact.

Plasticity of Bone.—Brailsford,[5] in a thought-provoking paper presented as a Hunterian lecture, describes molding of bones into deformity due to plasticity in paralysis, scoliosis, fragilitas ossium, rickets, Paget's disease, polycystic fibrous dysplasia, Perthes' disease and many other conditions. Quotations from Hunter clearly show that he realized that bones were plastic, even without roentgenologic studies. It is brought out that gravitational and muscular forces cause deformity, which is consequently seen much more frequently in the lower extremities.

Osteochondritis.—Two papers by Cole[6] and Doub[7] sum up present knowledge and conceptions of the various epiphysial lesions in their clinical and radiologic aspects. They do not include, however, the condition described as vertebra plana (Calvé).

Massaro[8] reports a case of vertebra plana with an eleven year follow-up and carefully differentiates it from Scheuerman's disease. The change is one of primary aseptic necrosis, usually of a single vertebra. The author prefers the designation vertebra plana to other names by which the condition is sometimes known. The destructive phase lasts about two months, and the regenerative phase is greatly protracted and may extend through a period of eight years or more. While vertebra plana may be descriptive of the late stage, it is probable that it is inappropriate for the earlier stages, just as coxa plana is descriptive of only a late stage of Perthes' disease.

Osteitis Deformans.—A paper by Dickson, Camp and Ghormley[9] constitutes a carefully worked out report on 367 patients with osteitis deformans. The authors have studied the roentgenograms of 200 of these patients. After noting the variations of architecture, they classify the disease into two phases, the sclerotic and the combined. The sclerotic phase is characterized roentgenographically by a homogeneous increase in density of bone, with detail of the cancellous trabeculae no longer perceptible. The combined phase represents nonhomogeneous alterations of density of bone represented roentgenographically by areas of osteo-

5. Brailsford, J. F.: Plasticity of Bone, Brit. J. Surg. **32**:345-357 (Jan.) 1945.

6. Cole, W. H.: The Clinical Diagnosis, Treatment and Prognosis of Epiphyseal Disturbances in Childhood, J. A. M. A. **127**:318-320 (Feb. 10) 1945.

7. Doub, H. P.: Aseptic Necrosis of the Epiphyses and Short Bones, J. A. M. A. **127**:311-317 (Feb. 10) 1945.

8. Massaro, A. F.: Vertebra Plana (Calvé), Radiology **45**:284-291 (Sept.) 1945.

9. Dickson, D. D.; Camp, J. D., and Ghormley, R. K.: Osteitis Deformans: Paget's Disease of the Bone, Radiology **44**:449-470 (May) 1945.

porosis, osteosclerosis and cysts. The paper accentuates other known facts about the disease. Familial incidence was observed. Twenty per cent of the patients had no complaints, the presence of the disease being discovered incidentally in the course of roentgenographic investigation. The most striking alteration observed in the blood of patients having osteitis deformans was elevation of the serum phosphatase level. Seventy-seven pathologic fractures occurred in the series, and sarcoma occurred in 3 instances. The authors state the belief, on a basis of their extensive study, that osteoporosis circumscripta is an early roentgenographic manifestation or precursor of osteitis deformans of the skull. Whereas earlier authors believed the skull and tibia to be the most frequent sites of the disease, this series reports the high incidence of 243 patients in 387 showing involvement of the pelvis.

Jacobs [10] describes a case of osteoporosis circumscripta cranii and expresses the opinion that it constitutes the early stage of Paget's disease. He feels that the condition should be looked for and a diagnosis made early in many instances on dental examination.

Seligman and Nathanson [11] report a case of Paget's disease with gross calcification of the soft tissues of the lower extremities. The patient received calcium and viosterol, but the authors express the belief that this was concomitant and not causal.

Hyperparathyroidism.—As a result of a deliberate attempt to improve diagnostic accuracy, Keating and Cook [12] state that 24 cases of hyperparathyroidism were discovered in two and one-half years at the Mayo Clinic, whereas only 14 cases were observed during the previous fourteen years. They emphasize that discovery of an increasing number of cases is entirely a reflection of a high index of suspicion and revision of the criteria necessary to make a diagnosis. Classic osteitis fibrosa cystica existed in only 7 of the 24 cases reported. In 9 there was mild or atypical disease of the bone, and in 8 no disease of the bone existed. They emphasize the frequency of calcification of the kidney or renal calculi, having found it in 92 per cent of their cases. An even more striking fact is that the chief clue to the diagnosis in 75 per cent of the cases was the presence of renal calculi. Prolonged study of the blood chemistry is often necessary to establish the diagnosis, which depends on the demonstration of an increase of calcium and a reduction of inorganic phosphorus in the serum as well as a reduction of

10. Jacobs, M. H.: The Jaws in Paget's Disease (Osteitis Deformans) with Special Reference to Osteoporosis Circumscripta Cranii, Am. J. Orothdontics (Oral Surg. Sect.) **31**:104-110 (Feb.) 1945.

11. Seligman, B., and Nathanson, L.: "Metastatic" Calcification in Soft Tissues of Legs in Osteitis Deformans: Case, Ann. Int. Med. **23**:82-91 (July) 1945.

12. Keating, F. R., Jr., and Cook, E. N.: The Recognition of Primary Hyperparathyroidism, J. A. M. A. **129**:994-1002 (Dec. 8) 1945.

calcium in the urine. The authors concluded by reemphasizing the conclusion of Albright and his colleagues that (1) hyperparathyroidism is commoner than generally supposed, (2) renal involvement is more frequent and diagnostically more important than osseous involvement and (3) every patient who has renal calculi should be suspected of having hyperparathyroidism until the contrary is clearly proved or until some other etiologic factor can be clearly demonstrated.

McClure and Lam [13] report 6 cases of hyperparathyroidism. Four patients were successfully treated by removal of adenomas. One died of acute parathyroid intoxication without the benefit of surgical intervention which had been advised. The other death occurred after operation on a patient in a hypoparathyroid state. As a result of their experience with these cases, the authors advise more frequent determinations of blood calcium levels in persons with symptoms of pressure on the nerve root, muscle pain and renal calcification and stones in order that a diagnosis can be made prior to the stage of osteitis fibrosa cystica or before spontaneous fractures have developed.

Fibrous Dysplasia of Bone.—There is currently a great deal of interest shown in fibrous dysplasia of bone, and numerous articles have appeared which add valuable information to the sum total of knowledge on the subject. Scholder [14] reports the case of a female patient followed over a period of eleven years, with relatively slight change in that period, presenting the diagnostic triad: precocious puberty, fibrocystic bone changes and cutaneous pigmentation. He advances a new hypothesis, that the syndrome results from a hypothalamic (pituitary) parathyroid disturbance.

Coley and Stewart [15] record the only 2 instances in the literature of malignant tumor of the bone occurring in fibrous dysplasia, thereby adding this condition to the group of diseases of the bone in which in certain instances malignant tumors of bone develop.

By far the most illuminating article on the subject is that of Jaffe.[16] He reemphasizes his earlier work, centering the disorder as a whole about the skeletal involvement, and his belief that the instances classified as Albright's disease are simply the most florid examples of the disorder as a whole. The author takes particular exception to the work of

13. McClure, R. D., and Lam, C. R.: End-Results in the Treatment of Hyperparathyroidism, Ann. Surg. **121**:454-469 (April) 1945.

14. Scholder, B. M.: The Syndrome of Precocious Puberty, Fibrocystic Bone Disease and Pigmentation of the Skin: Eleven Years' Observation of a Case, Ann. Int. Med. **22**:105-118 (Jan.) 1945.

15. Coley, B. L., and Stewart, F. W.: Bone Sarcoma in Polyostotic Fibrous Dysplasia, Ann. Surg. **121**:872-881 (June) 1945.

16. Jaffe, H. L.: Fibrous Dysplasia of Bone, J. Mt. Sinai Hosp. **12**:364-381 (May-June) 1945.

Thannhauser in 1944, which attempted to establish the thesis that fibrous dysplasia of bone is not an independent entity but that it is related by its clinical and histologic features to neurofibromatosis of Recklinghausen. Jaffe separates the entities and differentiates them clearly and convincingly. The article, to be properly appreciated, should be read in full.

Gaucher's Disease.—Tennent [17] describes what he refers to as a diagnostic osseous sign of this rarest of the three lipid storage diseases. The disease has been recorded in two forms, which may be referred to as visceral and osseous. The diagnostic points roentgenologically are found by study of the roentgenograms of the femurs. Well marked osteoporosis, which increases in degree from above downward, is an important sign. Expansion of the distal end of the femur is seen. There is found also a true over-all increase in breadth, but the diagnostic feature is a loss of the normal concavity above the medial femoral condyle. This is progressive, becoming a straight line and later a convexity. Cortical thinning and small medullary areas of erosion of trabeculae constitute the third sign.

Rickets.—Dunham and Thoms [18] report on a study of 10 children who had severe rickets in childhood and who were reexamined in adolescence. Five had rachitic pelves in adolescence, and 5 had nonrachitic pelves. As a result of their findings, they express the opinion that the older the child is when rickets is present, the greater the chance that pelvic deformity will occur. The entire group had, in adolescence, some deformity of the lower extremities. High ratios of sitting height to standing height were found in all but 1, which suggests retardation of growth of the lower extremities.

Scurvy.—Evans [19] reports a series of 93 cases. Sex distribution is about equal, and there was a striking limitation of cases to the latter part of the first year. Stages of the disease as it appears roentgenologically in order of their appearance are described and include ground glass type of demineralization, signet ring appearance of the epiphyses, submetaphysial notching, fragmentation or separation of the metaphysis and displacement of the epiphysis. Two important types of periosteal shadow are described; one appearing as a narrow triangular shadow, having its base at the metaphysis and extending up the shaft, and the other larger, more club shaped and extending along the greater length of the shaft. The differential diagnosis is discussed, including rickets, infection, trauma and neoplasm.

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17. Tennent, W.: Gaucher's Disease—The Early Radiological Diagnosis, Brit. J. Radiol. **18**:356-358 (Nov.) 1945.

18. Dunham, E. C., and Thoms, H.: Effects of Severe Rickets in Early Childhood on Skeletal Development in Adolescence, Am. J. Dis. Child. **69**:339-345 (June) 1945.

19. Evans, W. A., Jr.: Periosteal Lesions in Scurvy, Am. J. Roentgenol. **53**:147-156 (Feb.) 1945.

Phosphatase.—Silver and Golding [20] conducted experiments in vitro and in vivo to explain delay in healing of compound fractures, with particular reference to sulfonamide therapy in the wound. They found in maximal concentrations obtainable clinically in the blood that sulfanilamide, sulfapyridine and sulfathiazole produce no inhibition of bone phosphatase in vivo or in section. In maximal concentrations obtainable in tissues as a result of local application, sulfanilamide produces over 50 per cent inhibition of activity of bone phosphatase. Sulfapyridine and sulfathiazole, on the other hand, produce only slight inhibition. As a result of their experiments they suggest that sulfanilamide should not be used clinically as a local application in sites of fracture.

Calcification.—Vitamins, to some extent, at least, the "bread pills" of this generation, are generally looked on as being relatively harmless. Striking evidence of the potency of vitamin D in massive doses over a protracted period, however, is presented by Danowski, Winkler and Peters.[21] The authors present 2 cases which show that it is possible to produce calcification in soft tissues and renal failure. They express the opinion that the sequence of events is hypercalcemia, which increases the calcium content of the soft tissues, notably the kidneys, followed by renal calcification, which is the cause of renal failure. Fortunately, the process is reversible to some extent by discontinuance of the therapy.

Freyberg and Bauer [22] report a fatal case of vitamin D intoxication with metastatic calcification.

II. CONGENITAL DEFORMITIES

Prepared by

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Experimental.—For the past four years this review of congenital deformities has recorded additional contributions to this puzzling subject by Josef Warkany.[23] He points out that congenital malformations represent an important pediatric problem also, since they are a notable factor in infant mortality and in children's morbidity. Genetic, infectious

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From the Scottish Rite Hospital For Crippled Children.

20. Silver, P. H., and Golding, J. S. R.: Effects of Sulfonamides on Bone Phosphatase, Lancet **1**:528-529 (April 28) 1945.

21. Danowski, T. S.; Winkler, A. W., and Peters, J. P.: Tissue Calcification and Renal Failure Produced by Massive Dose Vitamin D Therapy of Arthritis, Ann. Int. Med. **23**:22-29 (July) 1945.

22. Freyberg, R. H., and Bauer, J. M.: Vitamin D Intoxication with Metastatic Calcification, Univ. Hosp. Bull., Ann Arbor **11**:61 (Aug.) 1945.

23. Warkany, J.: Congenital Malformations Induced by Maternal Nutritional Deficiency, J. Pediat. **25**:476-480 (Dec.) 1944.

and actinic factors have been proved to be etiologic principles leading to malformations. He has also shown that malnutrition of the embryo can be an etiologic factor.

It has been observed that animals fed poor diets gave birth to blind young. Warkany raised female rats on a highly purified diet free of carotene and vitamin A. The few rats able to terminate pregnancy produced young which were born blind and had deformed eyes. This is due to an arrest of development in an early embryonic stage. If vitamin A is added to the maternal diet, these deformities are not observed.

In previous articles on "Progress in Orthopedic Surgery" deformities have been listed which followed diet I. These were prevented by adding 2 per cent dried liver. The vitamin B complex was preventive. When the crystalline substances of vitamin B were tried, it was found that thiamine hydrochloride, nicotinic acid, pyridoxine and pantothenic acid, either alone or in combination, were ineffective. Riboflavin was found to be as preventive as liver. It was found that the aforementioned malformations could be prevented by administration of liver as late as the thirteenth day of gestation. He interprets this as proof that these malformations are not determined before the thirteenth day of prenatal life.

He says:

> Histologic sections show that the malformations are present already in cartilaginous structures, which indicates that the deviation from the normal development occurs before or at the stage of chondrification. Chondrification begins in the rat on the fourteenth and fifteenth days of fetal life. Thus, we can conclude that the malformations are determined not before the thirteenth day and not after the fifteenth day of gestation. A critical stage exists then in the 13- or 14-day rat embryo in which the presence or absence of sufficient riboflavin is of a decisive influence on the development of the embryo. At about this time most of the affected parts of the skeleton undergo rapid changes. Undifferentiated mesenchymal structures develop into the well-differentiated membranous skeletal elements which are the forerunners of the cartilaginous and osseous skeleton. This change from mesenchyme into the membranous skeleton seems to be impaired by a deficiency in riboflavin. The membranous skeleton exists only during a short period in prenatal life and therefore not much attention is paid to its morphology and physiology. But its constitution determines the fate of the permanent skeleton and its vulnerability deserves our special attention. It is of great interest to learn that riboflavin, a vitamin known to be necessary for normal growth, is also necessary for normal embryonic differentiation.

When he left the vitamin D out of diet I, he had a rachitogenic diet and the deformities were of a different pattern. The ulna, radius, tibia and fibula were decidely curved, and the ribs showed a peculiar angulation and broadening within their osseous parts. This pattern of malformation could be prevented when the maternal diet was supplemented by vitamin D.

Grebe [24] cites some statistics to demonstrate the incidence of congenital deformity. Thus Strohofer in 1933 reported an incidence of 0.5 per cent, Unterrichter in 1935 reported 1.3 per cent and Herrmann in 1939 reported 2.4 per cent of their admissions. Evidently congenital deformity is not rare. About one tenth of stillborn children are deformed and about one fifth of those dying within the first six months of life. After a discussion of the probability of further deformed offspring following the birth of infants with chondroplasia; syndactylism and polydactylism; cleft palate and harelip; cranioschisis, including anencephaly and spina bifida; malformations of the heart, and mongolism, the author concludes that none of these conditions justify sterilization, since the exact probability of recurrence has not been estimated. However, he suggests that the danger of producing abnormal offspring is greater when milder anomalies have already appeared in siblings. Some deformities are so slight that they would not affect the health. Severer deformities leading to prenatal death are less likely to be repeated in later offspring. In giving advice to prospective mothers one must consider not only the occurrence of deformity in siblings but also the condition of the general anlage, thus avoiding exclusion of good material because of slight defect.

Gregg [25] in 1941 found an unusual type of cataract in infants in Sydney, Australia. There was a remarkable similarity of the opacities of the lens, and a congenital defect of the heart frequently accompanied it. He found that all the mothers had had an attack of rubella during the first or second month of pregnancy. The health of the mother during the remainder of pregnancy was good. With the help of his colleagues 78 cases were collected. Some of these infants presented deaf-mutism. He refers to the work of Miss Mann, who showed that there was a relationship between a virus infection early in pregnancy and the appearance of certain congenital defects in the infant. She said that the most important determining factor in human beings for the effect of a maternally transmitted disease was the time of its action, only the cells which were in active division being affected. She showed that the time of the active division of the cells of the lens, internal ear and heart was at the time of the illness. The question is asked whether other diseases might not have a harmful effect on the developing embryo.

Greenthal [26] reports 2 additional cases from Milwaukee and gives references to others who have verified the fact that rubella during the

24. Grebe, H.: Angeborene Missbildung und weitere Kinder, Med. Klin. **39**: 484 (July 9) 1943.

25. Gregg, N. M.: Rubella During Pregnancy of the Mother with Its Sequelae of Congenital Defects in the Child, M.J.Australia **1**:313-315(March 31)1945.

26. Greenthal, R. M.: Congenital Malformations in Infant Caused by Rubella Early in Pregnancy: Report of Two Cases, Arch. Pediat. **62**:53-56 (Feb.) 1945.

first two months of pregnancy will cause deformities in infants. He wonders why similar cases have not been seen before and whether rubella has been severer during the last few years. He says:

> I believe that if rubella is the cause of such serious congenital malformations in the infant, then there should be no effort made to isolate children from rubella. I also agree with the suggestion that if a woman contracts rubella during the first two months of pregnancy, a therapeutic abortion is justified, as the chances of congenital defects affecting the infant are about one hundred per cent.

Adams [27] reports 2 new cases of patients showing both congenital cataracts and congenital defects of the heart. The importance of preventing rubella and other infections during the early months of pregnancy is pointed out.

Albaugh [28] has added 9 cases of congenital anomalies following maternal rubella in early pregnancy. He reviews the literature and analyzes the 78 cases reported. The commonest congenital lesions in the infants are cataracts, cardiac septal defects and patent ductus arteriosus, deaf-mutism and microcephaly. There was only 1 case of clubfoot reported. Nearly all the infants are poorly developed and present problems in feeding. The available data suggest that 100 per cent of the mothers who contract rubella in the first two months and approximately 50 per cent of those who contract it during the third month will give birth to infants with congenital anomalies.

In 1940 to 1942 congenital malformations were reported on 3,180 and birth injuries on 2,246 of the birth certificates filed in the state of New York, outside of New York city.[29] That in many cases malformations or injury were not recognized at birth or not reported by the attending physician is demonstrated by a comparison of the causes of death of children under 1 month with the statements on the corresponding birth certificates and likewise by a comparison with the Crippled Children's Register. On this basis a minimum estimate of malformations would be twice and birth injuries four times the reported figures.

Congenital malformation multiplies by at least 13 the risk of a child's dying in the first month of life and by 9 at 1 to 11 months. Birth injury raises the risk of death elevenfold during the first month but has no effect on the mortality of children aged 1 to 11 months.

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27. Adams, F. H.: Rubella in Pregnancy and Congenital Malformations, Journal-Lancet **65**:197-198 (May) 1945.

28. Albaugh, C. H.: Congenital Anomalies Following Maternal Rubella in Early Weeks of Pregnancy, with Special Emphasis on Congenital Cataract, J. A. M. A. **129**:719-723 (Nov. 10) 1945.

29. DePorte, J. V., and Parkhurst, E.: Congenital Malformations and Birth Injuries Among the Children Born in New York State, Outside of New York City, in 1940-1942, New York State J. Med. **45**:1097-1100 (May 15) 1945.

A case of blue sclerotics associated with a giant cell tumor in one eye of a Chinese schoolgirl was seen and reported by Chan.[30] Biomicroscopic studies as well as anatomicopathologic examinations of the enucleated eye showed thinning of the cornea, which was about three fourths as thick as that of a normal eye. Anatomic section demonstrated that the sclera was from one third to two thirds as thick as that of a normal eye, the ratio varying in different regions. Biochemical analysis of the sclera disclosed the presence of an abnormally large quantity of calcium. The author concludes, "It is believed that hyperfunction of the parathyroid glands may be a factor in the causation of this anomaly."

[Ed. Note (J. H. K.)—Blue sclera and brittle bones may be a rare condition in an eye clinic, but such cases are continually being seen in orthopedic clinics. They run in families and often through several generations. There is no evidence in this 1 case or in the reported cases that it is the result of a hyperparathyroid condition.]

A case of generalized neurofibromatosis accompanied with congenital deformities of the first three cervical vertebrae is reported by Peacock.[31] An operation was done to remove a tumor mass under the jaw. The patient died suddenly three days later, and autopsy findings, including the fusion of the first three cervical vertebrae, are given in detail. He says:

> The osseous conditions associated with generalized neurofibromatosis fall into two main groups. In one congenital skeletal defects occur. Kypho-scoliosis is frequently present, spina bifida and defects of the bony wall of the orbit are described. In the other, there are osseous manifestations directly due to the disease, exemplified by the results of periosteal lesions resulting in overgrowth of bony tissue and the enlargement of bones seen in various situations.

[Ed. Note (J. H. K.).—Many of the so-called congenital hypertrophies of parts or of the entire extremity are due to changes in the nerves to the part. There is an enlargement of the bone as well as of the soft parts. On careful examination patients with these conditions will show café au lait spots and when older neurofibromas.]

Brown and Pearce [32] wrote three papers on hereditary achondroplasia in the rabbit. The first has to do with the physical appearance. They summarize by saying that it is present at birth and is characterized by

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30. Chan, E.: Blue Sclerotics Associated with Bony Defects, J. Internat. Coll. Surgeons **8**:140-144 (March-April) 1945.

31. Peacock, A.: Developmental Abnormalities of Cervical Vertebrae in Case of Generalized Neurofibromatosis, Post-Grad. M. J. **20**:324-328 (Nov.) 1944.

32. Brown, W. H., and Pearce, L.: Hereditary Achondroplasia in Rabbit: Physical Appearance and General Features, J. Exper. Med. **82**:241-260 (Oct.) 1945. Pearce, L., and Brown, W. H.: Hereditary Achondroplasia in Rabbit: Pathologic Aspects, ibid. **82**:261-280 (Oct.) 1945; Hereditary Achondroplasia in Rabbit: Genetic Aspects; General Considerations, ibid. **82**:281-295 (Oct.) 1945.

reduction in size, by a disproportion of bodily parts, most striking in the extremities, and by an invariably lethal effect. The animals are stillborn or die shortly after birth.

In physical appearance and in the character of the skeletal changes as shown by roentgenograms, achondroplasia in rabbits has a remarkable resemblance to the disease in human beings and in cattle and dogs. The condition, which first occurred in offspring of purebred Havana rabbits is inherited. The mode of inheritance is on the basis of a simple recessive unit factor, and the appearance of nonachondroplastic transmitters (heterozygotes) is that of normal animals.

In the second paper they discuss the pathologic aspects.

At autopsy, the chief features of interest are: reduced size with disproportionately shortened extremities and large head, cutaneous and subcutaneous edema of variable degree and distribution, small shortened bones with a cartilaginous appearance and texture, immature teeth, and cleft palate in one-fourth the cases; blood-stained fluid in the thoracic and abdominal cavities; a comparatively small heart pointing to the right of the midline, a very large firm thymus, a large pale soft spleen, a large swollen liver with red mottling, and a stomach distended with thin greenish mucus but no milk.

The mean relative weights of all organs in terms of the net body weight were larger than those of normal new-born litter mates. The mean actual weights of the kidneys, the brain, and especially the spleen and the thymus were also larger than their respective normal values, those of the heart, liver, and adrenals were slightly smaller, while that of the pituitary was the same.

Histologically, all endochondral cartilages show marked abnormalities of differentiation with pronounced deficiency of ossification. Calcification of membranous bones is likewise deficient. The histological abnormalities of the long bones are very similar to, if not identical with, those characteristic of human fetal chondrodystrophy, the creeper fowl condition, the "bull-dog" calf, and achondroplasia of the dog.

No histological evidence was found in any organ which would suggest a basis for a responsible causal agent of the abnormality.

The third paper has to do with the genetic aspects and general considerations. It is the first instance of this abnormality in rodents to be reported. The variation arose in purebred Havana stock.

The abnormality is determined by the expression of a simple recessive unit factor, affected persons being homozygous for the factor. Females are somewhat more frequently affected than males, but the character is not sex linked. Rabbits heterozygous for the factor as determined by appropriate breeding tests have a perfectly normal appearance at birth and in later life.

The condition appears to be determined solely by the genetic constitution of the animal. Attention was drawn to the fact that, although the development of the achondroplastic form proceeds to birth at term, death regularly occurs at the time of or shortly after parturition.

Congenital absence of the odontoid process from the cervical axis has been reported in 1 case by Roberts and in 2 cases by Weiler. Scannell [33] adds another, that of a soldier who suffered sharp pain in the neck after a twist, sustained while wrestling. The pain was accompanied with generalized weakness, most noticeable in the arms. During the half-hour following injury, the weakness gradually disappeared. Roentgenographic studies, including laminagrams, revealed a complete absence of the odontoid process and abnormal mobility of the atlas on the axis in flexion and extension.

Allen [34] gives a good review of the literature and reports the findings in a Negro soldier who had engaged in strenuous infantry training for seven months without symptoms. He lists nineteen malformations in the upper part of the spine and thorax.

Ingersoll [35] presents a case of bilateral congenital high scapula with bilateral omovertebral bones which was treated by surgical removal of these structures, yielding functional improvement and giving some cosmetic benefit.

Grieve [36] reports 7 cases of congenital subluxation of acromioclavicular joint in the course of medical examinations in the air crew. In every instance the condition was bilateral. These cases show that mere displacement of the clavicle above the acromion need produce no disability.

A case of congenital recurrent dislocation of the head of the radius is reported by Bindman [37] because of its rarity and because it was recurrent and reducible. The case was that of a girl of 9 who had a dislocation of the head of the right radius while attempting to loosen a button on the left shoulder with her right hand. She felt a sudden pain in the right elbow and noticed a lump in front of the elbow, which disappeared on manipulation. The pain was momentary and not severe. Roentgenograms showed that the head of the radius was shallow and poorly formed.

Up to the present time some 24 cases of congenital humeroradial synostosis have been reported, nearly all of them in European literature. It is apparent that there is a hereditary tendency, since some have

33. Scannell, R. C.: Congenital Absence of Odontoid Process: Case Report, J. Bone & Joint Surg. **27**:714-715 (Oct.) 1945.

34. Allen, W. E., Jr.: Klippel-Feil Malformation: Report of Case in Adult, Radiology **44**:79-81 (Jan.) 1945.

35. Ingersoll, R. E.: Congenital Elevation of Scapulae with Bilateral Omovertebral Bones, New York State J. Med. **45**:1462-1463 (July 1) 1945.

36. Grieve, J.: Congenital Subluxation of Acromioclavicular Joint, Lancet **2**: 817-818 (Dec. 23) 1944.

37. Bindman, E.: Congenital Recurrent Dislocation of Head of Radius, Brit. M. J. **2**:354 (Sept. 15) 1945.

reported 2, 3 and even 4 such cases in the same family. Murphy and Hanson [38] report an infant born of normal parents who could not move his elbows, and the roentgenograms showed that there was no articulation at all at the elbow; the radius and humerus were one continuous bone.

Prof. Munir Ahmed Sarpyener, of Istanbul, Turkey, shows that many of the conditions which have been attributed to spina bifida are in fact due to congenital stricture of the spinal canal.[39] He says that it is a common phase of pathologic change that peripheral nerves are susceptible to pressure, whether this be the sudden pressure of a fractured bone fragment, the gradual pressure of exuberant callus or the slow envelopment of scar tissue.

> Thus far, there have been no references in the medical literature to congenital stricture not associated with spina bifida occulta. Studies extending over a long period have convinced me, however, that this pathological entity does exist, that it can be demonstrated by suboccipital injections of lipiodol, and that it is responsible for many hitherto unexplained clinical manifestations—enuresis, spastic or flaccid paralysis, and various deformities. . . .
>
> Just as tumors of the spine may cause spastic or flaccid paralysis, so congenital strictures may produce one or the other form of paralysis. The particular type is due not to the location of the lesion, but to the degree of pressure exerted on the cord.
>
> Thus far, I have been able to differentiate the following forms of congenital stricture of the spinal canal:
>
> 1. A narrowing of the canal, forming a ringlike constriction of the cord at one or more levels. This variety is usually found in cases of enuresis, and is readily cured by laminectomy. The relief of symptoms comes within a few hours of the operation. .
>
> 2. More extensive strictures, involving an entire region of the canal. This type is usually associated with spastic paralysis similar to that seen in Little's disease.
>
> 3. A localized stricture, causing compression of the spine and paralysis of certain groups of muscles. This is the type responsible for club-foot.
>
> 4. Atypical cases, causing a cleft either in the cord alone or in both cord and dura mater.

He presents many photographs and roentgenograms to show the remarkable improvement following laminectomy to relieve the pressure on the cord.

That the spinal canal may be greatly dilated as the result of an intraspinal neoplasm is well known, but that an equally pronounced dilatation may occur at any level as the result of a congenital anomaly of the cord structures is not generally recognized. Walker [40] reports

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38. Murphy, H. S., and Hanson, C. G.: Congenital Humeroradial Synostosis, J. Bone & Joint Surg. **27**:712-713 (Oct.) 1945.

39. Sarpyener, M. A.: Congenital Stricture of Spinal Canal, J. Bone & Joint Surg. **27**:70-79 (Jan.) 1945.

40. Walker, A. E.: Dilatation of Vertebral Canal Associated with Congenital Anomalies of Spinal Cord, Am. J. Roentgenol. **52**:571-582 (Dec.) 1944.

3 cases of myelodysplasia associated with dilatation of the spinal canal at the site of the anomaly. A case of vascular abnormality—angioma and aneurysm—of the spinal canal at the site of the lesion is also reported. The roentgenographic and pathologic manifestations of myelodysplasia are discussed. Although neoplastic conditions cause most instances of roentgenographically demonstrable enlargement of the spinal canal, congenital abnormalities of the spinal cord should be considered as etiologic factors.

A case of intrapelvic protrusion of the acetabuli is reported by Thoroughman [41] which is unique in that the hip symptoms are completely absent except for pain in the right knee. Otto originally described this condition in 1824, and only about 100 cases have been reported. It has been called Otto's pelvis, protrusion acetabuli and arthrokatadysis. These names should not be applied to deformities following trauma or metastatic processes. Neither should the conditions following infections be so classified. This condition probably results from a general disease in early life which has caused a softening of the bones. When osteoarthritis is seen, this is a later stage superimposed on a preexisting deep acetabulum. The intrapelvic protrusion is frequently asymptomatic until osteoarthritis occurs. The most constant physical findings are limitation of motion, particularly in abduction and external rotation. There is a rather constant flexion deformity with a compensatory lordosis. Therapy is unsatisfactory.

Mahaffey [42] reports the case of a soldier who had a fracture of the right lateral malleolus four years previously and who had continued to have pain in his foot. Roentgenologic examination revealed no space between the os calcis and the cuboid. A roentgenogram was made of the other foot, which had given him no trouble, and it showed the same.

[Ed. Note (J. H. K.).—Fusion of the calcaneocuboid joint is seen as the only deformity rarely and is frequently seen when associated with other deformities of the foot. This condition occurs much more frequently than the reported cases would indicate, because there is little reason for reporting congenital fusions.]

Veneruso [43] reports a soldier who had pain along the inner border of his right foot after prolonged walking or standing. He had only four toes on that foot. Roentgenograms revealed the complete absence

41. Thoroughman, J. C.: Intrapelvic Protrusion of Acetabuli, Am. J. Surg. **69**:238-242 (Aug.) 1945.

42. Mahaffey, H. W.: Bilateral Congenital Calcaneocuboid Synostosis: Case Report, J. Bone & Joint Surg. **27**:164-165 (Jan.) 1945.

43. Veneruso, L. C.: Unilateral Congenital Calcaneocuboid Synostosis with Complete Absence of Metatarsal and Toe, J. Bone & Joint Surg. **27**:718-719 (Oct.) 1945.

of one metatarsal and its phalanges and a fusion of the calcaneus and cuboid bones. No evidence of a joint between these bones could be found.

Matheis [44] states that good results can be obtained in almost all cases of congenital clubfoot if treated immediately. The inner structure of the foot and mechanical aspects are discussed. By utilization of growth pressure and delicate maneuvers tolerable even in newborn infants, a cure can be accomplished in a short time. The author describes a splint and cast. Treatment can begin on the day of birth, and cure is usually finished by the fourth month. No after-treatment is needed. The developmental cause of clubfoot is discussed. This is a false position of the supporting arch which in pathologic supination carries with it the great toe without changing its shape or position in relation to the arch. All other changes are caused by secondary lesions. Clubfoot in newborn infants is easily corrected by finger pressure, which may be applied in several sittings in cases of severe deformity, at intervals of two to three days. A plaster cast is applied after pronation, and the forces preventing a harmful effect will have a corrected effect. Once reduced by finger pressure the position is maintained by application of a splint and the foot fixed by strips of pasteboard to prevent lateral compression. The foot is placed in exaggerated abduction. Bits of rubber sponge are applied between the splint and leg and the transverse band and the external margin of the foot. Care must be exercised that traction and pressure do not counteract each other, with resulting injuries to the skin. The dressing is renewed every few days in a further corrected position. After overcorrection has been achieved a plaster cast is applied, the inner and outer margins of the foot being protected with cotton. The cast is applied to halfway up the thigh, the knee and thigh flexures being left free. Shortly before the plaster hardens, the foot is placed in the greatest possible position of overcorrection, with special attention to the heel. Shortly after hardening the cast is split. Both growth forces and movement are utilized as curative forces. A change is evident even after fourteen days. The cast should not be left on for more than three weeks, or too extreme pronation might result. After two or, at most, three weeks the thigh portion of the cast should be removed, and the knee is extended for one week to stretch the calf muscles. The cast is then reapplied in improved position. The thigh portion of the cast is removed from five to six times. When the foot remains in corrected position even against finger pressure, treatment is discontinued. Dorsal flexion and abduction following irritation of the sole prove that the peroneal muscle has recovered and that there is no danger of relapse. Six cases are reported.

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44. Matheis, H.: Die Sofortbehandlung des angeborenen Klumpfusses, Ztschr. f. Orthop. **74**:272, 1943.

The "medical treatment" of clubfeet is discussed in a rather theoretic article by Leonard.[45] He says:

> In medicine there are many occasions when there is a need for active treatment even though the mechanism which has produced the abnormal condition remains obscure. . . .
>
> It would seem that it should be possible to determine the exact age which is most satisfactory for successful treatment of a clubfoot by nonsurgical methods and that complete restoration of a fully functioning foot should be possible at that age in every case. . . .
>
> In other words, the failure is thought of as being the result of insufficient rather than wrong treatment. . . .
>
> If the process of ossification in the tarsal bones holds the clue to the problems of treatment of clubfoot and if the treatment is to be thought of as, first, a correction of the deformity and, second, a period of waiting for the elements of the foot to "set" in their proper shape like cooling plastic, the individual time schedule of ossification is of primary importance. . . .
>
> It may also be concluded that there is a certain percentage of abnormal delay in the appearance of ossification in those cases which have presented the greatest difficulty in the prevention of relapse. . . .
>
> An interesting corollary to the idea that delayed ossification effects the progress of recovery is a speculation regarding a possible relation between it and the etiology of clubfoot . . . [Clubfoot] appears . . . twice as often in boys as in girls. It has also been shown that the rate of ossification as a normal average in boys is slower by several months than in girls. A relative slowness of normal ossification is, therefore, found coupled with a relative greater incidence of clubfoot.

He uses an "ossification index," which is the year a given epiphysis usually appears. If there is a delay in the appearance of the center of ossification in the roentgenogram, the outlook is unfavorable and the foot should be held longer under treatment. If the appearance is normal, the prognosis is good and the foot will retain its correction. He expresses the opinion that a delay in the ossification of the tibial epiphysis may explain the inward torsion present in many of these cases. In cretins the delay in ossification is stimulated by the giving of thyroid. From work on rats he states the belief that the administration of thyroid should be accompanied with administration of anterior pituitary growth hormone.

He concludes:

> Medical treatment for the purpose of speeding ossification in a child with clubfoot through the period when the bones are being held in their normal position thus becomes a matter of giving adequate daily doses of thyroid substance with extra thiamine chloride which this medication demands and with the theoretical assistance of a diet rich in calcium, phosphorus, and vitamines A and D.

[Ed. Note (J. H. K.).—All who have treated clubfeet are familiar with the fact that some feet respond easily to corrective treatment and

45. Leonard, D. W.: Significance of Delayed Ossification in Treatment of Congenital Clubfoot, J. Pediat. **26**:379-389 (April) 1945.

others are difficult to correct and that these same conditions have a great tendency to recur. Frequently in bilateral conditions in which the rate of development might be expected to be the same in each foot, one foot will be more difficult to correct, even though it has had exactly the same treatment throughout. It is also known that there is a wide variation in the appearance of the centers of ossification for the bones of the feet. I have seen in clubfeet a center for the scaphoid at 2 years and have seen other untreated clubfeet in which it did not appear until 7. As soon as treatment was begun it made its appearance. I have studied grossly the time of development of the center of ossification in more than 500 patients with clubfoot and have not been able to deduct any rules of procedure. It is difficult to say whether a delay in ossification is the cause of the abnormality or the result of it. If a study of the roentgenograms of the feet shows that there is a slight delay in the appearance of ossification at 2, 3 and 4 years in the respective bones in boys, it is difficult for me to see how this can be responsible for twice as many boys having clubfeet, when this deformity becomes fixed early in the development of the embryo.]

III. TUMORS OF BONE AND OF SYNOVIAL MEMBRANE

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Osteitis Fibrosa Cystica.—Krigsten and Phalen [46] report 3 cases of fibrocystic disease of the femur in young soldiers. Treatment by means of curettage and bone graft gave satisfactory results. Pathologic fractures were the first evidence of the lesion in 2 of the cases. The authors state the opinion that cysts of bone arise as a result of localized congenital abnormality rather than as the result of hemorrhage. They distinguish two types of cysts of bone: the first, the fluid-filled, true cystic type lined by connective tissue and, the second, the fibrous "solid" type. They state the belief that the fluid type is more likely to occur near the metaphysis and the solid type in the shaft of a bone. They point out that a pathologic fracture occurring in the bone involved by a cyst

From the Section on Orthopedic Surgery, Mayo Clinic.

46. Krigsten, W. M., and Phalen, G. S.: Localized Fibrocystic Disease of the Femur: A Report of Three Patients Treated by Curettage and Bone Graft. Am. J. Surg. **67**:412-423 (Feb.) 1945.

always heals but that the cyst remains and another fracture may occur unless the cavity is packed with bone chips so that osteosynthesis takes place. The tibia and ilium were used as donor sites in their cases.

Goldthwait [47] describes a cyst of the sacrum that did not enlarge in thirty-five years. The patient was 44 years of age when she underwent an operation for removal of gallstones and uterine suspension in 1909. A cystic mass attached to the sacrum by a broad sessile base was noted in the hollow of the sacrum. Roentgenograms showed a large cyst which filled the sacrum, leaving only a shell of good bone at the periphery. The mass bulged posteriorly, as occurs in spondylolisthesis. The condition was treated conservatively with a lumbosacral support, and the patient got along well.

[ED. NOTE.—It is not uncommon to find cysts which have been present for many years without increase in size, but the cyst in the sacrum described by Goldthwait was present for a longer period than any that I know about. Pathologic fracture often is the first sign of a cyst. The cystic areas of decreased density with thinned and distended cortex are seen in the roentgenograms. The cysts commonly occur in young patients and are usually correctly diagnosed. I am convinced that when any doubt exists concerning such a lesion and fracture is imminent excision, biopsy and packing of the cavity with bone chips should be performed. The ilium or tibia provides excellent material for this.]

Metabolic Lesions.—Hand-Schüller-Christian Disease: Schairer [48] adds a report of 2 cases of eosinophilic granuloma to his cases reported in 1938. The first case was that of a girl 13 years of age in whom the tumor was located in the parietal bone and was removed surgically. The microscopic picture was typical, and recovery was uneventful. No evidence of recurrence was found two years later. There was no history of trauma in this case. The second case was that of a girl aged 4 years who had a similar tumor in the frontal bone.

The author also presents a summary of 15 cases reported in the literature, a macroscopic and microscopic description of the tumors and a discussion of prognosis and etiology.

Engelbreth-Holm, Teilum and Christensen [49] report 5 cases of so-called eosinophilic granuloma of bone in children 2 to 9 years of age. Clinically and morphologically a gradual transition to the fully developed Schüller-Christian syndrome was observed in these cases. Clinically the

47. Goldthwait, J. E.: A Case of Cyst of the Sacrum with No Increase After Thirty Years, J. Bone & Joint Surg. **27**:160-161 (Jan.) 1945.

48. Schairer, E.: Osteomyelitis mit eosinophiler Reaktion (eosinophiles Granulom des Knochens), Deutsche Ztschr. f. Chir. **258**:637-649, 1944.

49. Engelbreth-Holm, J.; Teilum, G., and Christensen, E.: Eosinophil Granuloma of Bone-Schüller-Christian's Disease, Acta med. Scandinav. **118**:292-312, 1944.

transition was from solitary eosinophilic granuloma to several foci to innumerable foci scattered throughout the skeletal system, together with diabetes insipidus, disturbance of growth and roentgenographic changes in the lungs. In several cases in which the initial finding was a solitary granuloma, continued observation revealed additional foci. The number of foci in the cases in the series increased with the period of observation. Morphologically, transitions were demonstrated from the lipid-free "eosinophilic granuloma" through granuloma with beginning lipid phagocytosis with Touton giant cells to entirely xanthomatous tissue. Eosinophilic granuloma no longer can be considered nosologically as an entity but must be considered as a not uncommon clinical monosymptomatic form of Hand-Schüller-Christian's disease, which often heals without becoming generalized. The histologic changes in these cases correspond exactly with the various histogenetic phases of Hand-Schüller-Christian's disease, including (1) the hyperplastic proliferative phase, (2) the granuloma phase, (3) the xanthoma phase and (4) the fibrous or healing phase. American writers have laid stress on differentiation of solitary eosinophilic granuloma from Hand-Schüller-Christian's disease, while German writers have emphasized the primary granulomatous nature of Hand-Schüller-Christian's disease but seem unacquainted with the clinical picture of solitary eosinophilic granuloma of bone. Ahlström and Welin, in Sweden, in 1943, were the first to suggest that the two were related. In some cases roentgen therapy and in others surgical therapy yielded good results. In still others no treatment seemed effective.

[ED. NOTE.—The two foregoing reports add to the literature 7 cases of Hand-Schüller-Christian's disease, or the so-called eosinophilic granuloma in children. This is an extremely rare disease of bones. These reports of clinical findings together with the microscopic and macroscopic studies add to knowledge of the disease so that accurate diagnosis can be made and removal of the tumor carried out. Roentgen therapy may be a useful adjunct to treatment. As more and more of these cases are encountered and the microscopic and macroscopic changes are studied, contributions are being made to the settlement of the question of transition forms. All cases of this disease should be reported.]

Benign Osteogenic Tumors.—Osteoma: Rankin [50] reports a case of a man 48 years of age in whom a slow-growing osteoma was present on the medial side of the lower part of the left femur, which caused impairment of circulation. The tumor had been present for thirteen years and had not increased in size during the last seven years but had gradually produced intermittent claudication of the calves of the legs after exercise. The leg exhibited cyanosis, venous distention and absence of dorsalis

50. Rankin, R. M.: Vascular Insufficiency of the Lower Extremity Due to Osteoma of the Femur, Surgery **17**:419-421 (March) 1945.

pedis and posterior tibial arterial pulsation. The roentgenogram revealed evidence of what later proved to be an osteoma of the medial lower part of the left femur with calcification of the femoral artery just proximal to the lesion. At operation it was found that the osteoma partially obstructed the femoral artery and vein at the outlet of the adductor canal. The vein was twice normal size below the obstruction. The arterial wall was calcified only in the portion just proximal to the obstruction. After the osteoma had been removed the patient obtained complete relief of the intermittent claudication and venous engorgement, but no change in arterial pulsation was observed.

[ED. NOTE.—This case, reported by Rankin, is unusual in that it concerns an osteoma, a benign tumor, which obtained sufficient size to cause obstruction of circulation in the leg. In this case the response to excision was favorable. In modern times osteomas are usually removed before they obtain a size sufficient to interfere with circulation.]

Fibroblastic Tumors.—Fibrosarcoma: Haagensen and Stout [51] review 95 previously reported cases and add 9 new ones. They state that synovial sarcoma is a rare, highly specialized form of malignant neoplasm of the extremities. In only 3 of the 104 cases did the patient obtain a five year cure. The tumor occurred in 3 males to every 2 females, and the average age of the patients was 32 years. The first symptom was pain, and nearly half of the tumors were located at the knee, although they have been reported from almost every joint in the extremities. The patients were treated by radical high amputation and dissection of the regional glands. Metastasis usually occurred by means of the blood, and the lungs were the most frequent site of metastatic involvement, although regional glands were involved occasionally.

Murray, Stout and Pogogeff [52] report that cultured tissue of synoviomas and normal synovia revealed the synovial cell as a specific type of cell, capable of considerable polymorphism. They further report that in tissue cultures of synovial sarcomas two types of cell and habits of growth prevail, although the distinction is less sharp in the tissue culture than it is in vivo. Histologic sections showed that two morphologically distinct types of cell coexist and display cancerous properties. In this respect synovioma exhibits certain similarities to the mesothelioma.

[ED. NOTE.—I have included synovial lesions in the fibrosarcoma group. It is my opinion that radical treatment is the method of choice in the majority of cases.]

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51. Haagensen, C. D., and Stout, A. P.: Synovial Sarcoma, Ann. Surg. **120**: 826-842 (Dec.) 1944.

52. Murray, M. R.; Stout, A. P., and Pogogeff, I. A.: Synovial Sarcoma and Normal Synovial Tissue Cultivated in Vitro, Ann. Surg. **120**:843-851 (Dec.) 1944.

Giant Cell Tumors.—Hughes [53] reports a case of giant cell tumor of the patella of a man 20 years of age. The patella had been enlarged for two and a half years after the patient was injured while playing football. Pain had developed after a blow across the knee received just prior to examination. The roentgenographic diagnosis was fibrocystic disease or giant cell tumor. The patella was excised, and the pathologist reported that the growth was a giant cell tumor. Recovery was uneventful.

Roemer [54] also reports a case of benign giant cell tumor of the patella. His patient was a man 28 years old whose knee had supposedly been injured. Roentgenographic examination disclosed a cystic defect of the patella. The upper half of the patella was excised, and the pathologic report stated that the lesion was a benign giant cell tumor.

Present [55] reports a case of subperiosteal tumor that developed two months after an injury. Tenderness was present, and supination of the left forearm was limited. Roentgenographic examination revealed a rounded, faintly calcified mass, 5 cm. in length, which rested on a saucer-like excoriation in the upper end of the right radius (questionable calcified hematoma). At operation an encapsulated tumor containing a mass of fleshy red material was removed. Pathologic examination showed that the tumor was composed of fibrous-appearing osteoid tissue with many giant cells and many pigment-laden macrophages (old hemorrhage).

[Ed. Note.—This condition, which probably resulted from ossification of a subperiosteal hematoma after injury, with pain and limited supination, gives a history suggestive of tennis elbow and demonstrates the value of roentgenographic study.]

Ribeiro [56] defines myeloplaxomas and suggests that they might originate in (1) myeloplaxes, (2) small mesenchymal cells, (3) lipid or xanthomatous cells or (4) "conjuntovascular structures." He brings up the questions of whether these can be considered to be tumors in the strict biologic sense and of whether they are of the same nature as giant cell tumor of the tendon sheaths. He describes the symptoms and clinical types and advocates treatment by means of simple curettage after biopsy and diagnosis. His opinion is that amputation and resection are indicated rarely, and he describes in detail a case of a girl

53. Hughes, J. E.: A Giant Cell Tumor of the Patella, Australian & New Zealand J. Surg. **14**:196-197 (Jan.) 1945.

54. Roemer, F. J.: Benign Giant Cell Tumor of the Patella, Am. J. Surg. **67**:563-566 (March) 1945.

55. Present, A. J.: So-Called "Subperiosteal Giant-Cell Tumor," Radiology **44**:77-79 (Jan.) 1945.

56. Ribeiro, T.: Os tumores de mieloplaxes dos ossos longos, e a sua cura pela enxertia óssea, Rev. españ. cir., traumatol. y ortop. **1**:181-204 (Sept.) 1944.

18 years of age in whom amputation was avoided by the use of osteoperiosteal grafts after curettage. The roentgenogram in this case revealed a cavity 4.5 by 3 by 2 cm. in the metaphysis of the femur and tip of the internal condyle. It was multilocular.

Jansson [57] presents a report of ten to twelve year cures in 4 cases of giant cell tumor treated by roentgen irradiation only. The lesions were located in the lower end of the femur or the radius. The patients were between the ages of 20 and 33 years. He states that low dosage of roentgen rays (500 to 1,000 r) given every second month until distinct ossification appears (three to four months) provides adequate treatment. He notes that after irradiation an initial period of increasing osteoclasis occurs before healing begins. One patient was cured by one dose of 600 r. In the past, bad results (dermatitis and ulcers) have been due to heavy dosage for this benign tumor. He expresses the opinion that the tumor recurs less frequently after irradiation than after curettage.

Hilton [58] presents 3 cases of giant cell tumor in which healing and recalcification occurred after roentgen therapy. Follow-up information was received from 1 patient for seven years and from another for eleven years. In 2 cases a diagnosis of sarcoma had been made elsewhere prior to treatment. Periosteal lipping and absorption of a large part of the cortex were evident on roentgenologic examination. The first evidence of healing after roentgen therapy was the reappearance or increase of the trabeculae. Hilton agrees with Jansson that the osteolytic type of giant cell tumor is only a more advanced form of the trabeculated type. After irradiation, in the initial period the tumor temporarily grows larger and more osteolytic and the bone is prone to fracture (one fracture in this series at the time of the report). This is followed by a period of healing. This author favors irradiation rather than surgical treatment of osteoclastomas.

Langenskiöld [59] states that giant cell tumors and ossifying hematomas are benign lesions which are oftenest incorrectly diagnosed as malignant lesions. He stresses the need for avoidance of unnecessary and useless amputations in cases of benign tumors, as well as the need for early treatment of patients with true osteosarcoma. He presents roentgenograms and details of 2 cases of giant cell tumor in which treatment consisted in scraping, insertion of 10 per cent solution of formaldehyde and then insertion of bone grafts. This author alines himself with Broca

57. Jansson, G.: Roentgen Treatment and Course of Cure of Giant Cell Tumour in Osseous System (10-12 Years' Cure), Acta radiol. **25**:569-579, 1944.

58. Hilton, G.: Radiotherapy in Osteoclastoma, Lancet **1**:110-112 (Jan. 27) 1945.

59. Langenskiöld, F.: Ueber Osteosarkomverdächtige, gutartige Erkrankungen, Acta chir. Scandinav. **87**:223-235, 1942.

and Foster and others who think that osteogenic sarcoma has a worse prognosis than is indicated in the literature and that apparent cures after radical operations are explained mainly by mistaken diagnosis.

Sacerdote de Lustig and Schajowicz [60] state that in cultures of the general group of giant cell tumors of bone the fibroblasts were more or less similar and the large giant cells persisted for a considerable period. Multinucleated cells were present at all times throughout the entire study of these cultures, and new giant cells were not formed. Due to the absolute absence of mitotic figures, these authors state that the cell division is amitotic. In the fragments of tissue examined great numbers of giant cells were observed in spite of the lack of osteoid and osseous tissue. This speaks against osteoclastic function of the giant cells. The authors are of the opinion that this tumor should not be considered to arise from osteoclasts.

Meyerding [61] presents a series of 40 cases of benign giant cell tumors treated by resection or excision and bone grafting. These cases were encountered in a period of thirty-one years, 1913 to 1943 inclusive. He reviews the history, the clinical, roentgenographic and microscopic findings and the follow-up data in all 40 cases. The diagnosis of benign giant cell tumor was confirmed by microscopic reexamination of tissue in all these cases. Massive resection and excision by curettage and cauterization appear to be safe methods of eradicating the tumor. The resultant cavities have been filled with autogenous bone, which stimulates rapid formation of bone, lessens the danger of collapse from fracture and tends to prevent deformity and disability. Meyerding expresses the opinion that excision of the tumor with bone grafting is a safe procedure in properly selected cases and that it tends to prevent deformity and disability. Illustrations show the distribution of the lesions in the skeleton. He presents 8 cases with roentgenographic studies.

[ED. NOTE.—The number of papers on giant cell tumor of bone illustrates the continued interest of the medical profession in this group of tumors. Treatment depends on the type, size and correct diagnosis of the lesion. Considerable difference of opinion exists as to the value of microscopic and roentgenologic diagnosis. I personally prefer that the roentgenographic diagnosis be confirmed by an expert pathologist, as the so-called clinical and roentgenologic features sometimes prove untrustworthy. If it is assumed that the roentgenologic diagnosis of giant cell tumor is correct and that the lesion may be cured by roentgen

60. Sacerdote de Lustig, E., and Schajowicz, F.: Cultivo de tejido de tumor gigantocelular de los huesos; contribución a la histogénesis de la célula gigante (nota previa), Rev. ortop. y traumatol. **14**:134-151 (Oct.) 1944.

61. Meyerding, H. W.: Treatment of Benign Giant-Cell Tumors by Resection or Excision and Bone Grafting, J. Bone & Joint Surg. **27**:196-206 (April) 1945.

therapy, the question arises of whether it is best to treat all tumors by roentgen therapy or whether excision and bone graft are wise in certain cases in which the lesion is situated so that they may be performed with a minimal loss of time and expense to the patient.

In none of the papers cited thus far was a case of malignant giant cell tumor reported. However, Phemister, whose paper is reported in the section on "malignant osteogenic sarcoma," includes 1 case. I have reported a series of malignant giant cell tumors, and in the nomenclature I have divided the giant cell lesions of bone into the benign giant cell tumor and malignant giant cell sarcoma. If these two terms are adhered to there will be less confusion concerning the diagnosis, prognosis and proper treatment of the patients. I think that it is impossible to distinguish the benign giant cell lesion from the malignant giant cell sarcoma by roentgenographic examination alone.]

Vascular Tumors.—Pohle and Clark [62] report a case of multiple hemangiomas involving the skin, brain, vertebrae, ribs, long bones of the extremities and the pelvis, in which the patient responded well to roentgen therapy. The widespread lesions of the bones recalcified or regressed. The total observation period in the case was seven and a half years.

Brunner [63] discusses the diagnostic difficulties encountered in cases of Ewing's sarcoma and reports 5 of his own cases. The lesions in the 5 cases were located in the humerus, tibia, femur, tenth rib and fifth cervical vertebra. The ages of the patients varied from 10 to 32 years. It is Brunner's opinion that only on rare occasions is it possible to distinguish Ewing's sarcoma from osteomyelitis, especially if biopsy is not performed. This differentiation is especially difficult when the lesion is located in the vertebrae and the flat bones. The author expresses the opinion that the therapeutic test in which deep roentgen rays are used is not decisive, that the roentgenologic features are not diagnostic or that the characteristic periosteal reaction is not a reliable sign. Based on the roentgenograms made early in the course of the disease, the diagnosis usually was chronic osteomyelitis, and only after repeated roentgenologic examination at intervals of three to four weeks was the presence of a malignant lesion suspected. In the majority of cases the blood picture was normal. Slight leukocytosis occurred only in 1 case. The sedimentation rate was considerably elevated in 2 of the cases and slightly in 1. Decided elevation of temperature was not

62. Pohle, E. A., and Clark, E. A.: Multiple Cavernous Hemangiomata of the Skin, Brain and Skeleton: Report of Case Treated by Roentgen Rays and Observed for 7½ Years, Urol. & Cutan. Rev. **49**:283-287 (May) 1945.

63. Brunner, W.: Das Ewing-Sarkom (Retikulosarkom des Knochenmarks) mit besonderer Berüksichtigung seiner Differentialdiagnose auf Grund von 5 eigenen Beobachtungen, Deutsche Ztschr. f. Chir. **258**:540, 1944.

present. Although a single symptom cannot be pathognomonic, the early pain and especially the night pain are the best leads. Only 1 of the 5 patients died. In that case the lesion was in the tibia. The patient with the lesion of the rib was treated by radical excision of the tumor followed by irradiation, the other 3 patients received irradiation only. When roentgen therapy was given, swelling of soft tissue and pain disappeared within a few weeks. Restoration of the bone, however, took considerably more time.

Reeves [64] reports 2 cases, 1 of his own and the 1 of Archer, in which primary round cell tumors resembling Ewing's sarcoma were present in the mandible and rib respectively. In each case roentgenographic evidence suggested that pulmonary metastatic lesions were apparently cured by irradiation. The primary tumors were eradicated by excision and irradiation therapy. The first patient, a girl 9 years of age, had a lump in the left mandible, which was removed by excision, and roentgen therapy was given. The growth recurred several times (1936, 1938 and 1941) and was treated surgically (excision of the lymph nodes in the neck because of metastasis and removal of the left mandible in 1941), and further irradiation was given. In 1942 multiple pulmonary metastatic lesions were given deep roentgen therapy and cleared completely. At an examination in 1944, no metastatic lesions were present in the lungs and the patient was apparently in good health. Archer's patient was a girl 6 years of age who was given deep roentgen therapy for multiple pulmonary metastatic lesions from a lesion which was apparently primary in the right sixth rib. (Biopsy of the rib had revealed sarcoma.) In March 1943, eleven years later, examination showed no evidence of the lesion nor any symptoms. The lungs were free of involvement. Thus, in 2 cases of seemingly hopeless conditions cures apparently were obtained.

[ED. NOTE.—Vascular neoplasms of bone are more responsive to roentgen therapy than are other tumors of bone, and successful roentgen therapy is practically diagnostic of Ewing's sarcoma in cases in which other findings support the diagnosis. A lesion of this type may disappear entirely, and the patient may remain in good health for many years. I have reported 1 case, that of a girl who was 11 years old on her first admission, in which treatment consisted in roentgen therapy and administration of Coley's toxins. She was alive and well more than seventeen years after treatment. The lesion was located in the left femur, and biopsy revealed that it was an endothelioma. If it is possible, it is preferable to excise and irradiate some vascular tumors. However, some are of such size and so located that roentgen therapy is the only practical form of treatment. It should be remembered that these

64. Reeves, R. J.: Round Cell Tumor of Bone Resembling Ewing's Tumor, South. M. J. **38**:302-306 (May) 1945.

small round cell lesions of the Ewing type are not infrequently interpreted to be inflammatory, or a diagnosis of osteomyelitis is made even after operation and microscopic examination.]

Malignant Osteogenic Sarcoma.—Phemister [65] reports 2 cases of sarcoma of the femur. The first patient, a man 26 years of age, underwent resection of part of the lower part of the femur and upper end of the tibia. The diagnosis was malignant giant cell tumor. A graft, 11 inches (27.9 cm.) in length, from the tibia was cut in two and placed so that it bridged the gap left when the sarcoma was removed; the grafts were placed in both the coronal and the sagittal plane and were fixed above and below by means of threaded wires. The second patient was a girl 11 years of age who had a recurrent cartilaginous tumor in the shaft of the lower part of the right femur, which was considered to be a chondrosarcoma of low grade. Resection of about 6 inches (15.2 cm.) of the shaft of the femur was performed, the knee joint being left intact. Tibial grafts were inserted as described in the first case. Excellent results were obtained in both cases; both grafts were gradually transformed into tubular bone which resembled the missing part. Both patients are living and well approximately four years after operation.

Sarasin [66] emphasizes the complexity of roentgenologic images of tumors of bone, which renders diagnosis difficult, and the great importance of careful clinical examination and of the history. He describes a case in which the patient was a child. Three months after the patient had bronchopneumonia a hard swelling developed in the upper half of the left femur. Roentgenographic examination revealed destruction of bone in the upper third of the femur; the picture suggested the presence of sarcoma, and operation was advised. The child's mother, however, insisted that the swelling was the result of the injections given the child for the pneumonia, and a second roentgenographic examination revealed evidence of inflammation. Puncture yielded pus, and on culture Koch's bacilli were identified. In this case operation would have had fatal results. Sarasin emphasizes that differentiation of Brodie's abscess is impossible by roentgenographic examination and that biopsy is an important measure. He describes various types of sarcoma and is of the opinion that roentgen therapy plus operation is the treatment of choice and that Ewing's sarcoma is often present for long periods before diagnosis is made. He calls attention to the disproportion between the roentgenographic image and the microscopic extension of the tumor; occasionally an enormous tumor will effect only slight roentgenologic changes. Sarasin also points out that histologic studies are important

65. Phemister, D. B.: Rapid Repair of Defect of Femur by Massive Bone Grafts After Resection for Tumors, Surg., Gynec. & Obst. **80**:120-127 (Feb.) 1945.

66. Sarasin, R.: Les tumeurs osseuses; leur diagnostic et leur traitement, Praxis **33**:530-534 (July 27) 1944.

in identifying myeloplax tumors. He expresses the opinion that these tumors are benign. Surgical or roentgen therapy may be used, but he states the belief that some of the lesions do not respond to treatment as readily as others and that if the cyst resolves with formation of slightly vascular bone tissue the course is longer. Even though myeloplax tumors are not malignant they should be treated as such because failure of an initial attempt will greatly impair the results of later treatment. If roentgen therapy produces no change, the tumor probably is an atypical myeloplax tumor which is not radiosensitive. It is his opinion that total extirpation is indicated, and he reports 2 illustrative cases. The tumor may be transformed to sarcoma.

Metastatic Tumors.—Cohn and Cohn [67] report 4 cases in which the patient had pain low in the back and was treated for sacroiliac strain by means of sacroiliac belts, diathermy, heat and other measures, when the correct diagnosis was small primary carcinoma of the breast (proved microscopically) with skeletal metastasis. In 3 cases evidence of metastatic osseous lesions was found on initial roentgenographic examination, but in 1 of the 4 negative results were obtained until four months thereafter. They advised repeated roentgenograms. They discuss the route of metastasis by the way of Batson's mechanism through the valveless vertebral veins from the thoracoabdominal veins by coughing, straining, lifting and other means. They are of the opinion that breasts of all patients should be examined carefully for tumors, especially when pain low in the back or pelvic pain is present, and that pain in the lower part of the back may be the first evidence of neoplastic disease.

Peck [68] reports 2 cases in which the patient had pain low in the back from metastatic prostatic carcinoma. After orchectomy pain was relieved completely and roentgenographic evidence of healing of the metastatic lesions was observed. Reports in the literature of 300 cases of carcinoma of the prostate were reviewed. In 75 per cent of these 300 cases pain was relieved and the serum acid phosphatase level dropped, often within twenty-four hours after orchectomy or administration of estrogen. Peck states that carcinoma of the prostate with metastasis is the only condition in which the level of serum acid phosphatase is high and quotes from the report of Dean and his associates as follows: "In their studies they used the Bodansky unit, in which 0.7 unit was considered normal and any value over 1.0 was considered pathognomonic of metastases from carcinoma of the prostate The normal alkaline phosphatase, using this method, is 1.5 to 5.0." Orthopedic surgeons must be constantly on the lookout for this condition in any man more than

67. Cohn, T. D., and Cohn, H.: Low-Back Pain as the Presenting Symptoms of Malignant Breast Tumors, New England J. Med. **232**:342-343 (March 22) 1945.

68. Peck, R. I.: The Treatment of Skeletal Metastases Secondary to Carcinoma of the Prostate, J. A. M. A. **127**:17-19 (Jan. 6) 1945.

50 years of age who has pain low in the back, and rectal examination as well as routine orthopedic examination should be made. When doubt exists after roentgenographic examination for evidence of metastasis, determination of the value for serum acid phosphatase will aid in the diagnosis and the results of later determinations will be indicative of the efficacy of treatment.

Dargent and Hutinel [69] describe the removal, by block dissection, of an atypical epithelioma of the right lobe of the thyroid. The superior thyroid vein was found to be invaded by the growth. Even though roentgenographic examination revealed malignant destruction of the fourth thoracic vertebra and the patient had paraplegia, operation was performed and roentgen therapy given. The patient gradually became able to walk after this. Follow-up information was obtained concerning this patient for two years. At last examination, the Babinski sign was still positive but motor function was good. The bone had not healed, as far as could be determined from the roentgenographic appearance. Evidently the tumor was extremely radiosensitive. These authors also cite the case of a woman who underwent surgical removal of the manubrium sterni for metastasis from an epithelioma of the urinary bladder. After she remained well for thirteen years, clavicular metastatic lesions were found.

Ringertz and Ehrner [70] report 2 cases of neurofibromatosis with sarcomatous degeneration. The first was a case of generalized neurofibromatosis in which the patient was a woman 52 years of age. A fibrosarcomatous tumor had been removed from the sciatic nerve on the right side when she was 21 years of age. Thirty-one years later the tumor recurred in both sciatic nerves, and death occurred five months after the second operation. Postmortem examination revealed fibrosarcoma of the thorax, liver, vertebrae and right arm. The second patient was the son of the first patient. He underwent operation at the age of 19 years for fibrosarcoma of the neck. Three years later the tumor recurred locally and rapidly increased in size in spite of intensive irradiation. The patient died of bronchopneumonia at the age of 24 years. This patient, who was apparently free from neurofibromas of the skin, appeared to have extensive neurofibromatosis of practically all the spinal nerves. Review of the literature showed that 105 proved cases of neurofibromatosis with sarcomatous degeneration have been reported. Apparently sarcomatous changes occur in about 13 per cent of the

69. Dargent, M., and Hutinel, J.: Epithélioma atypique du corps thyroïde. Mètastase vertébrale et paraplégie. Lobectomie thyroïdienne élargie et radiothérapie. Résultat au bout de 2 ans, Lyon chir. **37**:374-379, 1941-1942.

70. Ringertz, N., aand Ehrner, L.: Ueber Sarkombildung bei Recklinghausenschr Neurofibromatose (mit Beschreibung zweier neuer Fälle), Ztschr. f. d. ges. Neurol. u. Psychiat. **176**:297, 1943.

cases of neurofibromatosis. These sarcomatous changes show a high frequency of location in the nerve plexuses of the extremities. Sarcomatous degeneration rarely occurs in cutaneous neurofibromas. The authors state that operation in cases of neurofibromatosis tends to produce a malignant reaction. Metastasis, especially to the lungs, pleura, liver and skeleton, occurs rather frequently.

Trafton and Perkin [71] present a report correlating the results of nine hundred and twenty-five determinations of the level of serum acid phosphatase by the modified Bodansky method at the Lahey Clinic. They bring out that acid phosphatase is present in significant amounts only in the prostate gland and that the level in the serum is abnormally high only in cases of carcinoma of the prostate with metastasis. When the level of serum acid phosphatase is 0.8 to 1 unit per hundred cubic centimeters and the level of serum alkaline phosphatase is normal, the presence of carcinoma of the prostate with metastasis is indicated. Values of 1.2 or more units are pathognomonic. The authors consider the average normal values in males to be 0.5 unit and in females 0.42 unit. The level of serum acid phosphatase was elevated in 24 of 50 cases of prostatic carcinoma, the level of alkaline phosphatase in 22 and the levels of both in 17. In 81 per cent of the cases in which metastasis to bone had occurred an initial elevation was noted. The highest value was 34 units. The level of serum acid phosphatase was elevated in 3 cases before roentgenograms revealed metastasis. Castration resulted in a pronounced fall in the serum acid phosphatase level in 24 cases, and the level did not rise for from one to three years. Clinical improvement occurred. The determination of the phosphatase level is of diagnostic and prognostic value and is a guide to endocrine therapy.

[ED. NOTE.—The reports of these authors emphasize the importance of thorough study of the history and of the clinical and laboratory observations and roentgenograms in all cases of unexplained pain, since metastasis may be the principal cause of pain.]

Classification of Bone Tumors.—MacCarty [72] presents his theory of origin of neoplastic tumors evolving from hemorrhage in bone marrow or under the periosteum. He states that many, if not all, osteogenic sarcomas if viewed early would present the picture of giant cell tumor resulting from hemorrhage. He considers that a second group of bone tumors arises from endothelial cells and a third group from the bone

71. Trafton, H., and Perkin, H. J.: The Clinical Significance of Serum Acid Phosphatase with Especial Reference to Carcinoma of the Prostate Gland, Lahey Clin. Bull. **4**:59-63 (Oct.) 1944.

72. MacCarty, W. C., Sr.: The Genesis, Nomenclature and Classification of Bone Tumors, in Thomson, J. E. M.: The American Academy of Orthopaedic Surgeons Presents Lectures on Peace and War: Orthopedic Surgery, Ann Arbor, Mich., Edwards Bros., Inc., 1945, pp. 248-256.

marrow cells. On that basis he proposes a classification based on (1) tumors composed of adult differentiated tissue cells, (2) tumors composed of young regenerative or partially differentiated cells, (3) tumors composed of mixed cells which may or may not be differentiated and (4) tumors composed of undifferentiated cells. He states the belief that the primitive cell of bone is probably an endothelial cell which is closely related to fibroblasts.

Miscellaneous.—Beard [73] reports his study of the effects of penicillin and choline on Emge sarcoma transplants in three groups of rats. Each rat in the first group (control) received two Emge sarcoma transplants. Each rat in the second group received the same type of transplants and in addition an injection of 2 cc. of a solution containing 333 Oxford units of penicillin daily. Each rat in the third group received the same type of transplants and 40 mg. of choline chloride daily in the drinking water. Beard states that penicillin and choline apparently caused a significant number of the tumor transplants to fail to grow, and the tumors that did grow were, on the whole, smaller when penicillin had been used. He feels that no conclusions are justified in regard to the number of takes and the slower growth of the tumors due to the many factors that may influence these characteristics of tumors.

[ED. NOTE.—While no justifiable conclusions were reached in this study, such experiments should be continued, as it is possible that they may lead to discovery of a means of controlling or inhibiting the growth of malignant lesions.]

Gratzek, Holmstrom and Rigler [74] present a review of 1,363 cases in which malignant lesions of the pelvis were irradiated. They found 25 cases in which the patient had damage of bone that included aseptic necrosis, sclerosis, absorption and fractures. From 1936 to 1941 a high dose of irradiation was given in 568 cases, and bone sequelae developed in 3.2 per cent of this group. After repeated roentgenographic examinations it was concluded that pain preceded sclerosis and necrosis and that fractures occurred later. The time after irradiation before the damage was detected was between five months and five years. In 13 cases of this series the femoral neck was found to be fractured, and 55 cases in which this had occurred were reported in the literature up to 1941. Three cases of multiple fractures of the pubis, ischium, ilium and sacrum are reported in detail. In their study the authors found that in patients with postirradiative changes the lesions of bone healed readily

73. Beard, H. H.: The Effect of Penicillin and Choline upon the Appearance, Growth and Disappearance of the Emge Sarcoma in Rats, Exper. Med. & Surg. **2**:286-289 (Nov.) 1944.

74. Gratzek, F. R.; Holmstrom, E. G., and Rigler, L. G.: Post-Irradiation Bone Changes, Am. J. Roentgenol. **53**:62-76 (Jan.) 1945.

with the usual orthopedic treatment. They state that the lesions result from irradiation fibrosis and obliteration of the blood supply to the bone, and they warn physicians not to mistake these lesions for metastatic lesions and give more irradiation. Additional irradiation was given in 1 case, and damage was increased.

[ED. NOTE.—These postirradiation changes in bone are of great importance and should be looked for in cases in which roentgen therapy has been given and fractures have occurred.]

(To Be Continued)